Ovarian abscess and tubal abscess. (Page 243.)

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OF

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ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PEDIATRICS, OBSTETRICS, GYNECOLOGY, ORTHOPEDICS, PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY, OTOLOGY, RHINOLOGY, LARYNGOLOGY, HYGIENE, AND OTHER TOPICS OF INTEREST TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION THROUGHOUT THE WORLD

EDITED BY

A. O. J. KELLY, A.M., M.D., PHILADELPHIA, U.S.A.

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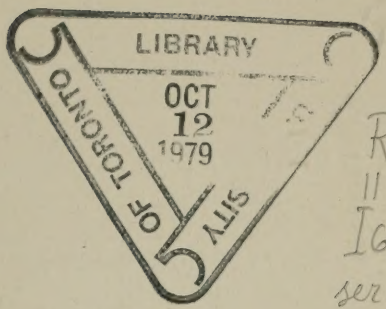

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CONTRIBUTORS TO VOLUME III

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ABRAHAMS, BERTRAM, M.B., B.Sc., F.R.C.P., Assistant Physician to the Westminster Hospital; Joint-Lecturer on Medicine in the Westminster Hospital Medical School; Examiner to the Conjoint Board, London, England.

BANTI, G., M.D., Professor of Pathologic Anatomy, and Instructor in Clinical Medicine and General Pathology, in the University of Florence; Chief Physician to the Hospital "Santa Maria Nuova," Florence, Italy.

BRADFORD, E. H., M.D., Professor of Orthopedic Surgery in the Harvard Medical School; Visiting Surgeon to the Children's Hospital, Boston, Mass.

CARMICHAEL, E. SCOTT, M.B., F.R.C.S.(Edin.), Assistant Surgeon to the Royal Hospital for Sick Children; Gynecologist to the Royal Public Dispensary, Edinburgh, Scotland.

CLOGG, H. S., M.S.(Lond.), F.R.C.S.(Eng.), Assistant Surgeon to the Charing Cross Hospital, and Surgeon to the Evelina Hospital for Children, London.

CORNER, EDRED M., M.A., M.B., B.C.(Cantab.), B.Sc.(Lond.), Senior Assistant Surgeon to the Hospital for Sick Children, Great Ormond Street; Surgeon to Out-Patients, St. Thomas's Hospital, London.

CUMSTON, CHARLES GREENE, M.D., Surgeon to the Floating Hospital, Boston.

GALLANT, A. ERNEST, M.D., Professor of Gynecology in the New York School of Clinical Medicine; Consulting Surgeon to the Jamaica Hospital, New York; Assistant Gynecologist to the Roosevelt Hospital Out-patient Department, New York; Formerly Special Assistant in the Department of Surgery (Professor W. W. Van Arsdale) of the Good Samaritan Dispensary, New York City.

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HILL, EDWARD C., M.D., Professor of Chemistry in the Medical and Dental Departments of the University of Denver, Denver, Colorado.

KATZENBACH, WILLIAM H., M.D., Professor of Medicine in the New York Polyclinic Hospital and School for Graduates in Medicine, New York City.

LERMOYEZ, MARCEL, M.D., Physician to the Paris Hospitals.

M'Gregor, A. N., M.D., F.F.P.S.G., Assistant Surgeon to the Glasgow Royal Infirmary, Glasgow, Scotland.

Miller, D. J. Milton, M.D., Physician to the Episcopal Hospital of Philadelphia.

Patton, Joseph M., M.D., Professor of Diseases of the Chest in the Chicago Polyclinic; Clinical Professor of Diseases of the Chest in the Medical Department of the University of Chicago, Illinois.

Pinard, A., M.D., Professor of Obstetrics at the Paris Faculty.

Pirie, J. H. Harvey, B.Sc., M.B.(Edin.), Medical Officer to the Scottish National Antarctic Expedition of 1902–4.

Potts, Barton H., M.D., Rhinologist and Otologist to the German Hospital, Philadelphia.

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Soupault, Maurice, M.D., Physician to the Paris Hospitals, Paris, France.

Stevens, A. A., M.D., Professor of Therapeutics and Clinical Medicine in the Woman's Medical College of Pennsylvania, and Lecturer on Physical Diagnosis in the University of Pennsylvania, Philadelphia.

Thomas, T. Turner, M.D., Assistant Surgeon to the University and the Philadelphia General Hospitals; Assistant Instructor of Surgery in the University of Pennsylvania.

Wainwright, John W., M.D., New York.

Watkins, Thomas J., M.D., Clinical Professor of Gynecology in the Northwestern University Medical School; Gynecologist to St. Luke's, Mercy, and Wesley Hospitals, Chicago, Illinois.

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Treatment

THE TREATMENT OF ACUTE PLEURISY

BY A. A. STEVENS, M.D.

Professor of Therapeutics and Clinical Medicine in the Woman's Medical College of Pennsylvania, and Lecturer on Physical Diagnosis in the University of Pennsylvania, Philadelphia

Pleurisy, like other forms of serositis, has a diverse etiology. While the disease is always infectious, the nature of the infection and the conditions under which it occurs are variable. It follows, therefore, that no one method of treatment will be suitable in every case. In the present paper it will be sufficient to consider the best means of dealing with that large group of cases, formerly regarded as primary or idiopathic, in which the disease develops more or less acutely in persons who are otherwise apparently healthy.

As a rule, the attack develops shortly after some unusual exposure, but here, as in pneumonia, the chilling probably acts no more than as an auxiliary cause. Recent investigations have shown that a very large proportion of the cases of fibrinous or serofibrinous pleurisy, occurring in adults and usually ascribed to catching cold, are in reality of tuberculous origin. By resorting to animal inoculation, Le Damany proved that no less than 86 per cent. of 55 cases of serous pleurisy, which formerly would have been classed idiopathic, were tuberculous, and in the same manner Eichhorst, Grober, Aschoff, Netter, and others have also demonstrated the close relationship between the two diseases. Judging from these reports and the evidence afforded by clinical observation, it may fairly be assumed that at most not more than 20 per cent. of the cases of serous pleurisy are non-tuberculous, or due to pneumococcic, streptococcic, or staphylococcic infection. This fact cannot be too strongly emphasized, as it

has a most important bearing not only on the prognosis of the disease, but also upon its treatment.

No matter how mild the attack of pleurisy, rigid hygienic treatment must be insisted on from the beginning. The patient should go to bed and remain there until the fever has entirely disappeared and the exudate shows signs of receding. The sick-room should be well ventilated but free from drafts, and the atmosphere kept at an even temperature of from 65° to 68° F. The food should be nutritious and easily digestible, but preferably dry, especially after the development of the effusion.

The first indication for special treatment is usually to relieve the pain in the side. Morphin hypodermically is useful, but its effect, even in large doses, is temporary. A far more reliable remedy is the application of a few wet cups or leeches. This measure, the value of which is well recognized but too often forgotten, affords immediate and permanent relief in the vast majority of cases without the aid of opium. Dry cupping is not nearly so effective. After the local bleeding thin poultices may be applied to the chest if necessary. I have not been very favorably impressed with the heavy, unwieldy cataplasms of fuller's earth and glycerine which lately have been so much vaunted for their supposed depleting effects, and cannot regard them as a satisfactory substitute for the time-honored poultices of flaxseed meal. Some practitioners prefer ice-bags to poultices, but in my experience cold applications have not often been tolerated in simple pleurisy. Strapping the affected side from mid-spine to mid-sternum with broad strips of adhesive plaster, as originally suggested by Frederick T. Roberts, is very helpful in the mild attacks of pleurisy that so frequently develop in the course of ordinary phthisis, but I have rarely found them sufficient to relieve pain when it is severe.

Unfortunately we have no very potent means of checking the progress of the inflammatory process, once it has begun. Among drugs, tartar emetic, aconite, quinin, and calomel have in turn won the confidence of the profession, but they have all gone the way of asclepias, which, owing to its fancied efficacy in acute inflammatory diseases of the chest, has long been known as pleurisy-root. The only drug which seems to have any special virtue is salicylic acid. This remedy has been warmly advocated by Aufrecht, Fiedler, and,

in this country, by Dock, but its value is limited. I have found it chiefly useful in sthenic cases occurring in robust persons and accompanied by sharp pain and high fever. When such cases are seen early sodium or ammonium salicylate, in doses of a dram or a dram and a half (4 to 6 grams) in the twenty-four hours, sometimes relieves the pain, lowers the temperature, and apparently exerts a modifying influence on the inflammatory process itself. In other cases, and these constitute the great majority, the drug is absolutely useless.

After the acute symptoms have subsided the indications are to accomplish the removal of the fluid, to maintain nutrition, and to secure complete expansion of the lung. Very little can be done in the way of hastening the absorption of the exudate by medicinal treatment. Counterirritation by means of flying blisters appears to have a good effect. In vigorous subjects the administration of saline purgatives, according to the method suggested by Matthew Hay, is sometimes of service. The object of this treatment is to deplete the blood of its serum and thereby favor the abstraction of fluid from the lymph spaces. The liquid consumed by the patient is restricted to about two-thirds of the amount of urine that he passes and every morning or every other morning from ½ to 1 ounce (15.0 to 30.0 grams) of epsom salt is given, in as little water as possible, an hour before breakfast. If for any reason epsom salt is not well borne compound jalap powder, in doses of from 30 to 40 grains (2 to 2.6 grams), may be substituted. Diuretic remedies, such as digitalis, caffein, and theobromin, may be tried, but they are far less effective than purgatives. Diaphoretic agents should not be employed. They are not only without influence on the inflammatory exudate, but, when used repeatedly, they are likely to cause considerable depression. Potassium iodide has long enjoyed some reputation as an absorbent in serous effusions, but it is of very doubtful efficacy. For a number of years I prescribed it faithfully in every case of simple pleurisy, but as I did not observe any effect from its use, except at times irritation of the stomach or the unpleasant symptoms of iodism, I have long since ceased to recommend it.

Under medicinal treatment, although doubtless often independently of it, very slight pleural effusions frequently disappear spontaneously in the course of one or two weeks. When the effusion is

considerable, however, say up to the fifth rib, treatment by drugs usually proves extremely unsatisfactory and not uncommonly several months elapse before the chest is freed of liquid. The more of such cases I see, the more I am impressed with the idea that there is but one treatment for pleurisy with effusion, and that is aspiration. While it is quite true that serous pleurisy itself rarely proves fatal, and that in the large majority of cases resorption of the fluid ultimately occurs, much saving of time is effected by early resort to paracentesis. A few years ago Delafield reported 200 cases of serous pleurisy treated by aspiration. Of the whole number, 182 patients left the hospital cured. In more than one-half of the cases the duration of the disease before the operation was between ten and thirty days. The length of time which elapsed from the aspiration to the entire cure of the patient was within one week in 64 cases, within two weeks in 138 cases, within three weeks in 158 cases, within four weeks in 169 cases, and within six weeks in 179 cases. Thus it will be seen that in more than three-fourths of the cases recovery occurred within two weeks.

If the pleurisy were the only condition to be considered, the shortening of the patient's stay in his home or in the hospital would in itself be ample justification for the forced evacuation of the exudate by operative means. We know, however, that in most cases pleurisy is not an independent affection, but a secondary process developing in the course of latent tuberculosis, and the earlier the patient can be gotten up and out into the open air the greater will be his chances of permanent cure. It is for this reason, especially, that early intervention is to be recommended. I believe it is a good rule, therefore, not to wait longer than a week in any case, unless the effusion is very slight and shows definite signs of receding. The presence of fever is not a contraindication; indeed, the temperature often falls upon the removal of the fluid. Irrespective of the period of the disease paracentesis is urgently demanded: (1) When there is sufficient fluid in the pleural sac to excite severe dyspnea, cyanosis, persistent cough, or failing pulse; and (2) when the fluid reaches the level of the third rib and there is marked displacement of the neighboring organs.

Aspiration is rarely attended by accidents of any kind. Sudden death, the result of cerebral anemia, has been reported; but the cases

in which this accident has occurred during tapping are more than offset by those in which a fatal termination has resulted directly from increased intrathoracic pressure. In a few instances the intercostal artery has been wounded. Norman Moore [1] has recently reported a case in which death resulted from this cause. Another serious and even fatal complication of thoracocentesis, but also extremely rare, is a peculiar form of pulmonary edema, which is manifested by cough, intense dyspnea, and profuse albuminous expectoration. According to Riesman,[2] who has studied 32 cases from the literature, the principal cause of this condition is either too rapid or too great a withdrawal of fluid. With ordinary care the risk of transforming a serous pleurisy into an empyema is very slight. Griffiths [3] carefully studied 151 cases in which paracentesis was performed, and found that in two cases only did the fluid become purulent. In one of these cases the pleurisy was complicated with erysipelas. Occasionally attempts at aspiration are unsuccessful. The cause of failure may be plugging of the cannula, great thickening of the pleura, or encapsulation of the effusion. Under these circumstances it may be necessary to make several trials before a flow can be established.

Before each operation the apparatus should be thoroughly examined and tested, especial care being had that the stop-cocks are properly closed. If this precaution were always observed many annoying experiences at the bedside would be avoided. The patient should be brought to the edge of the bed, placed in a semirecumbent position, and supported by an assistant. The most favorable site for the puncture is usually in the sixth or seventh intercostal space between the mid-axillary line and the angle of the scapula. When the amount of effusion is very moderate, however, it may be desirable to choose a point on the posterior aspect of the chest. It is scarcely necessary to add that the operation should be done under strict antiseptic precautions. Local anesthesia may be secured by means of a spray of ethylchoride or the application of a block of ice that has been dipped in salt. The needle should be inserted with a quick thrust along the upper margin of the rib, the depth of the

[1] Lancet, June 10, 1905.

[2] American Jour. of Med. Sciences, April, 1902.

[3] British Medical Journal, vol. i, 1887, p. 831.

puncture being gauged by the forefinger. After the needle has been introduced, the operator should satisfy himself that it is freely movable, should hold it in position throughout the operation, and as the evacuation proceeds should slowly raise the exposed end so as to keep the inner opening below the level of the fluid in the pleural sac. The aspiration should be effected slowly, and at intervals it should be stopped by compressing the conducting tube. Too rapid evacuation may excite syncope, or cause engorgement of the lung and edema. The amount of fluid that should be removed will depend upon the size of the effusion and the ease with which it can be evacuated. Even with large effusions it is rarely necessary to withdraw more than a liter, and under no circumstances should extreme efforts be made to obtain the largest possible amount of fluid. The operation should be terminated at once if persistent cough, severe pain, dyspnea, palpitation, tendency to syncope, or other untoward symptoms appear. When the requisite amount of fluid has been removed, the needle should be withdrawn quickly from the chest, and the puncture closed with adhesive plaster or collodion. If the fluid reaccumulates, which is not infrequently the case, paracentesis should be repeated after the lapse of a week. In Delafield's cases the operation was done once in 142 cases, twice in 45 cases, three times in 9 cases, and four times in 4 cases.

While the immediate prognosis of pleurisy is good, the remote prospect is always cause for anxiety. V. Y. Bowditch [4] investigated the subsequent history of 90 patients treated for pleuritic effusion by the elder Bowditch from 1849 to 1879. He found that 30 had died of tuberculosis and at least one of those still living was tuberculous. Barrs [5] reported 57 cases treated at Leeds from 1880 to 1885, and of this number tuberculosis developed in 21. Salanoue-Ipin,[6] of the French navy, ascertained that of 301 patients with pleurisy 84 afterward became tuberculous. Hamman [7] got reports from 88 of 172 patients treated at the Johns Hopkins Hospital, and of these 88 at least 30 later became tuberculous. In view of these facts the necessity of keeping under prolonged observation every

[4] Cited by Whitney, Twentieth Century Pract. of Med. vol. vii.
[5] Brit. Med. Jour., Aug. 10, 1890.
[6] Arch. de Méd. Navale, No. 4, 1900.
[7] Cited by Osler, Practice of Medicine, 1906, p. 644.

patient who has had serofibrinous pleurisy, and of urging him to adopt such measures as will promote his general nutrition, is obvious. Open-air living, an abundance of wholesome, easily-digested food, tonics like iron and arsenic, and exercises intended to bring into play the muscles of respiration, are to be recommended. In short, the after-treatment should be precisely that which is laid down for incipient tuberculosis.

THE TREATMENT OF CERTAIN FORMS OF BRONCHITIS

BY JOSEPH M. PATTON, M.D.

Professor of Diseases of the Chest in the Chicago Polyclinic; Clinical Professor of Diseases of the Chest in the Medical Department of the University of Chicago, Illinois

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The treatment of bronchitis is generally undertaken almost entirely from a symptomatic standpoint, the remedies employed being selected according to the amount and character of the sputum and for the relief of local irritation as expressed by cough. There is thus no disease, in all probability, in which routine prescribing is more often practiced than in bronchitis, favorite empirical methods being employed without definitely locating the seat of the trouble or determining the exact nature of the infection. That these inexact methods are relatively successful cannot be disputed, for a large proportion of bronchial infections are of the upper portion of the bronchial tract, their most prominent feature is a tendency to self limitation, and local irritation is a marked clinical feature of their course. Symptomatic medication, therefore, is successful in most instances. Moreover, these infections of the upper bronchial tract are due to a great variety of organisms, and, excepting possibly the influenza bacillus, it is difficult to place the responsibility of any particular organism.

In the finer divisions of the bronchial tract, however, we find inflammations of a more or less distinct type, of a somewhat definite etiology, with a distinct tendency toward localization, and exhibiting a subacute clinical course bordering on chronicity, if not actually chronic. Again, these inflammations may manifest a type of symptoms so acute as to be deceptive, except for the duration of the clinical course.

The etiology of these forms of bronchitis rests in a more or less definite infection. The particular organisms involved are streptococci, staphylococci, and influenza bacilli. Pneumococci are often

present in small numbers, but generally have no special relation to the bronchitis. The three organisms first mentioned may be associated in a given case, but the type of the disease will vary according as the one or other predominates.

Localization, especially in the more chronic types, is in the posterior portion of the lower lobes. The lateral and anterior portions of these lobes are less often affected. The middle lobe of the right lung may be occasionally involved, while the upper lobes are rarely affected. These distinctly local affairs may be associated with very limited areas of lobular pneumonia.

In *streptococcic bronchitis* the area involved is likely to be more extensive than with the other types of infection. The disease may also be bilateral, or more than one lobe of one lung may be affected. The clinical type may be much like that of acute pneumonic tuberculosis,—a fever of 102° F. in the afternoon, pulse of 110 to 120, night sweats, loss of flesh, cough which is usually harsher and more troublesome than that of tuberculosis, considerable mucopurulent sputum, but less exhaustion than would be expected in tuberculous cases in the absence of pneumonia. The sputum contains streptococci and usually some staphylococci. This form of bronchitis may be associated with general streptococcic blood infections, which are frequently fatal. The duration under treatment may be from three to five months.

In *staphylococcic bronchitis* there is a marked tendency to localization. The topography is essentially the same as in the former type, except that the local tendency restricts the majority of instances to a portion of one lobe, and it is seldom bilateral. The course is subacute or chronic, and the clinical symptoms less marked. The temperature will be from 99° to 100° F. in the afternoon. The pulse is rarely above 90. The cough is less constant and harassing and is more or less periodical. The sputum is alternately frothy and heavy, and contains staphylococci almost entirely, though influenza bacilli may be present. Night sweats are absent. There may be no loss of weight. The clinical features may resemble those of the first stage of phthisis. The course may be from two to three or four months.

Influenzal bronchitis may be general or local. When local we usually find a free admixture of staphylococci. Infections which

are more purely influenzal are prone to affect the upper tract and to be more or less general in character. The clinical symptoms vary with the portion of the bronchial tract involved, and lobular pneumonia is a frequent associate of influenzal inflammations of the smaller bronchial tubes.

Differentiation of these forms of bronchitis, especially of the first, from tuberculous conditions may be difficult and at times only possible through careful examination of the sputum. The topography of these inflammations is a strong point in their differential diagnosis, as they are nearly always basic in situation when localized, and even if more or less general, or bilateral, they seldom affect the apical regions—which are so prone to tuberculous involvement. Nevertheless, the ubiquitous character of the tuberculous lesions should cause us to be ever alert to the possibility of either primary or secondary involvement by tuberculous processes of these portions of the lungs.

In the treatment of these forms of bronchitis there are two important points to be considered: First, that the expectorants so freely used in the treatment of ordinary forms of bronchitis are practically useless for the relief of the inflammations under consideration. They affect chiefly the upper portion of the bronchial tract and are popular elements in most every cough medicine for the relief of acute forms of bronchitis, but for subacute or chronic forms of inflammation of the finer bronchial tubes, especially those of a localized character, they appear to accomplish nothing.

The second point is that cough sedatives are inadmissible, except in isolated instances. The general principle governing this point is whether the cough is beneficent or not, and if so, whether it is accomplishing its object without causing undue disturbance. The utilitarian purpose of cough is to free the air-tract from some irritation, but its efficiency is not always proportionate to its specific indication. It, therefore, may create more trouble than it cures and may require to be held in check. This is particularly true of inflammation of the upper bronchial tract, where irritation causing cough arises from the hypersensitive state of the bronchial nerves, more than from obstructive conditions from which the air-tract might be freed by the act of coughing. This portion of the bronchial tract, especially at the bifurcation of the trachea and of

the main and medium-sized bronchi are sensitive areas, while the finer divisions of the bronchi are comparatively devoid of sensation. Mucus accumulating in the finer tubes, therefore, will not cause cough until it has become detached and works its way along the bronchus to one of these sensitive points. Thus in nonacute bronchitis of the finer tubes we have periodical cough, often in the morning upon rising, when the mucus has been forced out of the smaller bronchi during the night, reaches the sensitive portion of the tract with the first movements of the patient, and coughing results. These coughing spells may occur once or twice a day and are nature's effort to free the air-tract from obstruction and irritation; they should not be interfered with unless the accompanying irritation is so severe or prolonged as to render the cough more persistent than is desirable from a beneficent point of view.

In the more acute and extensive forms of bronchitis it will be necessary to employ some sedative for the cough, but in a general way we may say that the indications for cough sedatives diminish directly as the inflammation is of chronic tendency, as it involves the finer tubes, and as it exhibits localizing features. Nothing, perhaps, will better illustrate this feature of the matter than the experiences of patients who have taken the various patent and proprietary cough medicines to be found upon the market. They are invariably made worse and more uncomfortable, dyspnea is added to their troubles, and they come to the physician after a few weeks of self-medication with disordered stomachs and a history of restless nights and of periodical coughing which renders them miserable. We should, therefore, be careful not to thwart nature's attempts at relief in these cases, and unless her efforts can be aided by the judicious administration of sedatives they should be withheld.

Self-limitation of inflammatory processes in the smaller bronchi is not nearly as marked as in the larger tubes. Symptomatic medication, therefore, is relatively less successful, and empirical remedies are more frequently failures than in the management of acute bronchitis. The therapy of the localized forms of bronchitis demands the use of some agent which is eliminated by the mucus of the bronchial tract, which will not produce much irritation of the tissues during the elimination, and which will, if possible, exert

some tonic influence upon these tissues. All these effects may be obtained by the use of iodine.

Iodine is one of the most diffusible substances in the blood. It can be recovered from the saliva within fifteen minutes after its administration. Aside from its elimination by the kidneys it leaves the body mainly by way of the mucosa of the respiratory tract. If it is true, as claimed, that at the points of elimination of iodine by the mucous surfaces the chemical changes set free ozone which causes what irritation ensues, it is possible to obviate any such effect by the selection of proper preparations, and by correct dosage. The alterative and tonic effect of iodine is a matter of every-day clinical experience. Over thirty years ago, Bouchard ascertained that an increased nitrogeneous metabolism resulted from the administration of iodides, and Duchesne has called attention to the increased excretion of urea from the use of iodides. Whether the tonic effect is simply the reaction of the system when some handicap has been removed, as has been claimed by some syphilographers in relation to the action of iodine in specific disease, or whether the tonic action is the result of a more or less maintained dilatation of the finest blood-vessels resulting in improved nutrition, as has been claimed by certain French therapeutists as resulting from the iodine element of the various salts, are matters of theory. The clinical facts of improved nutrition and elimination cannot be denied, and clinical results determine in the end the value of any remedy.

Iodides increase waste and the elimination of the products of waste, and if given in too large doses for too long a time may cause emaciation and depression of vital forces. Again, individual susceptibility to iodine varies more, almost, than with any other drug. Therefore, preparation and dose are very important. The observations of the authors above mentioned show that with the iodides of sodium, ammonium, and calcium, increased nitrogenous elimination is not maintained for so long a time after their administration ceases as is the case with the potassium salt. There is, therefore, less secondary depression from their continued use, as is also the case with the iodide of strontium.

Whether we accept the observation of Benedict regarding the paralysing influence of the iodide of potassium on the cardiac and

respiratory nervous system of frogs, as of practical value or not, the majority of authorities concede that this salt is more depressing to the heart than is the sodium preparation; and when continued administration of even small doses for a period of two or three months is necessary—as is the case in the management of the affections under consideration—it is obvious that the preparation employed is worthy of as careful attention as are the idiosyncracies of the individual to be treated.

Iodide of sodium is, perhaps, the most generally-useful preparation of its class because of its acceptability to the stomach, because it shows little tendency to cumulative effects, and because it does not cause so much or as lasting depression. When stimulation is desired the ammonium salt may be more suitable, while with nervous patients the strontium preparation often acts best. In patients with neurotic tendencies or asthmatic manifestations a motor depressant may be advisable, and here grindelia robusta may be advantageously combined with any of the iodides. A somewhat routine prescription in my clinic is the following:

Sodium iodide	2 drams	8|0
Fluid extract of grindelia robusta	1 ounce	30|0
Syrup of licorice	6 drams	22|0
Simple elixir, sufficient to make	3 ounces	100|0

Mix. S. One teaspoonful (5 c.c.) three or four times daily.

When dyspnea of spasmodic occurrence is a feature, the fluid extract of euphorbia pilulifera may be substituted for the grindelia robusta in this prescription.

In elderly patients with general or a bilateral form of bronchitis, the following will often be of service:

Potassium iodide	2 to 3 drams	8 to 12|0
Antimony and potassium tartrate	1½ grain	0|1
Tincture of belladonna	1 dram	4|0
Compound syrup of sarsaparilla, sufficient to make	3 ounces	100|0

Mix. S. One teaspoonful (5 c.c.) three times daily.

In feeble patients when cough exhausts them the ammonium salt may be used in this mixture. With patients who do not take these mixtures well, or are susceptible to iodides, we may use the

iodides in the form of the tablets of iodonucleoids, which are well borne by the stomach. With sensitive persons we may use the syrup of hydriodic acid, a remedy which is also very useful with children.

In anemic subjects the syrup of the iodide of iron will, at times, be found almost a necessity, and many persons, especially at or after middle life, whether anemia is present or not, will find little benefit resulting from the iodides, unless at the same time we administer arsenic. The following is an eligible formula:

Potassium or sodium iodide	2 to 3 drams	8|0 to 12|0
Solution of potassium arsenite	1 to 2 drams	4|0 to 8|0
Simple elixir	3 ounces	100|0

Mix. S. One teaspoonful (5 c.c.) three times daily.

If the digestive system is irritable the strontium salt may be substituted in this prescription with advantage.

Another agent useful in all forms of subacute or chronic bronchitis is gaduol. It would be useless to enter into a consideration of the chemistry of this so-called extract of cod liver oil or discuss the question of its being an extract, or, as has been claimed, a ptomain product. These questions are not material in this connection in view of the results obtained by its administration. It is of particular utility in children with whom it is so difficult to secure the continuous administration of any suitable remedy for any required length of time. Gaduol does not disturb the stomach and has no unpleasant effect. It may be given in capsule, worked up with some suitable excipient, but the best method for its administration is in the from of wine, of which the following is a useful formula, agreeable alike to children and adults:

Gaduol	64	grains	4|25
Alcohol	1	ounce	30|00
Fuller's earth	4	drams	15|00
Peptonized manganese	1½	drams	6|00
Water	2	ounces	60|00
Sugar	2	ounces	60|00
Angelica wine, to make	16	ounces	500|00

The gaduol is mixed with the alcohol and triturated thoroughly with the fuller's earth. The manganese is dissolved in the water,

to which is added the sugar and wine, and the whole is then thoroughly mixed. This mixture should stand for a day or two, being occasionally agitated. It is then filtered and enough wine is added to preserve the volume. The dose is a tablespoonful three or four times a day. As far as the alterative effect is concerned this preparation is equal to cod liver oil, while it is infinitely more agreeable. Its effects in the forms of bronchitis under consideration are better than those of cod liver oil.

In bronchitis due to the streptococcus or the influenza bacillus the irritation of the upper respiratory tract may be sufficient to necessitate sedative measures. Morphin, heroin, or codein may be required, but frequently the use of these may be obviated by occasional deep inhalations of a spray from the following mixture:

Menthol	20 grains	1|30
Powdered camphor	15 grains	1|00
Oil of eucalyptus	15 minims	1|00
Benzoinol, to make	3 ounces	100|00

This may be used with any air-pressure apparatus or with a Davidson's No. 59 hand atomizer. Spells of spasmodic coughing at night may often be controlled by inhaling a few drops of ethyl iodide from a bottle or handkerchief. This substance is easily decomposed and should be kept in a tightly-corked bottle which should be either colored or well wrapped.

When fever is a feature of these types of bronchitis, and it is likely to be with streptococcic infections, even if they are quite local, it is not advisable to give active febrifuges. The temperature may be modified from one to one and one-half degrees by giving every three or four hours, two or three grains of acetanilid combined with one or two grains of quinin sulphate or quinin bromide. This will not depress the patient. Night sweats, when they occur regularly, may be treated with atropin ($^1/_{60}$ grain, 0.001 gram) at bedtime, camphoric acid (5 grains 0.3 gram) in capsule, at 5, 7, and 9 o'clock, or agaricin ($^1/_{10}$ grain, 0.006 gram) at 2 P.M. and 7 P.M.

Climatic treatment is not to be neglected in those cases which do not respond to treatment promptly. Certain localities in the central states, especially in the region of the great lakes, are more

or less unfavorable to the treatment of these types of bronchitis. Fresh air and sunshine are as necessary here as in other forms of pulmonary disease. We should be careful about allowing patients with a degree or more of fever to go away from home surroundings. They should not be allowed to go into high altitudes when in this condition. Climatic treatment should be regarded as a measure for the convalescent stage. Cases of a chronic type with a tendency to recurrence during the winter season often do best in low altitudes and warm, moist climates, such as Jamaica, Bermuda, Porto Rico, Cuba, or the gulf coast of Florida. Others who require a fairly dry climate do well in central or western Texas, or in Mexico.

Our results with these patients will usually be in proportion to the careful attention and individual discrimination with which we manage them.

THE TREATMENT OF DILATATION OF THE HEART

BY WILLIAM H. KATZENBACH, M.D.

Professor of Medicine in the New York Polyclinic Hospital and School for Graduates in Medicine, New York City

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It is so often said that very little can be done for organic conditions of the heart that students and young practitioners of medicine very readily imbibe the idea that cardiac therapeutics is not very promising. Any one, however, who has had considerable experience with heart cases will surely have learned the precious lesson that, as the heart is the hardest worked organ in the body, so too it is the one most capable of compensating for its own defects; that it is the organ which most readily and promptly yields to properly applied therapeutic measures, being possessed of powers of recuperation quite beyond expectation in many cases.

Most young physicians are likely to think that if a patient present symptoms of distinct dilatation of the heart with the apex-beat well outside the nipple line, with the heart sounds inclined to be irregular and rather distant, and with a distinct murmur present —the prognosis is almost inevitably unfavorable, and only a relief of symptoms for a time can be afforded. This is, however, entirely too discouraging a view to take of these cases of dilatation of the heart: many of them respond promptly to proper treatment, with an eventual recovery of regularity and strength of heart action that makes them among the most satisfactory cases to treat.

Dilatation of the heart may be acute or chronic; it is especially acute cases that I wish to discuss. Chronic dilatation of the heart usually means a very serious condition of the heart tissues, with degenerative changes in the heart muscle. After all, it is on the heart muscle that the eventual prognosis depends, and degenerative changes, of course, cannot be eradicated. Even in these cases, however, the toning up of the muscular fibres that are as yet undegenerated brings about an improvement in heart action and, as a consequence, in the general condition of the patient, that is quite sur-

prising. Such improvements may be much more lasting than was originally expected by the physician, and serve very well to illustrate the heart's wonderful recuperative powers.

Acute dilatation of the heart, however, is in quite another order of disease, and amelioration of the condition may be confidently expected under favorable conditions. The patients usually come complaining of certain subjective sensations that are a source of great annoyance. Usually there is a feeling of crowding within the chest, as if there were not room there for all the organs. To this is added the consciousness of the heart's movements, especially after meals or when the patient attempts to lie on the left side. Besides these sensations of subjective discomfort dyspnea usually develops and may become quite a marked feature. Patients often have to use a number of pillows in order to sleep at all. They often have a dread of going to bed because of the difficulty of breathing and the sense of impending death that is likely to come with it.

These symptoms, while they occur more frequently in persons beyond middle life, may be noted in patients who are comparatively young. Certainly it is not a rare occurrence to find them in individuals under forty years of age. Usually the first symptoms have been ushered in by some over-exertion. In New York City the most frequent story is that the patient was hurrying for an elevated train, saw one approaching his station, ran up the stairs as fast as possible in order to catch it, and then as he reached his seat in the cars had a distinct attack of rather severe discomfort in the precordial region with considerable palpitation and shortness of breath. Sometimes, however, the attack comes on only as the result of over-exertion when the individual is very tired, though there has been no special strain put upon the heart by rapid movements.

In most of these cases a systolic murmur may be heard at the apex of the heart, a murmur which is conducted toward the left and may sometimes be heard even at the angle of the scapula. It is never a harsh murmur, but is always soft in character except in cases in which there is a history of rheumatism or some other infectious disease complicated by endocarditis which brought about a serious crumpling in the tissues of the mitral valve. As a matter of record in many of these cases there is no history of rheumatism, and in spite of the fact that not only is a murmur present but the

apex-beat is displaced, it is very doubtful whether a genuine valvular lesion exists, since after rest and proper care and treatment the murmur disappears and the apex-beat resumes its normal location.

The only way to make this part of the subject clear seems to be to illustrate it by a typical case. This is not meant to be a wonderful example of therapeutic efficacy, nor an exceptional instance of cardiac recuperation, but an example of what can be accomplished by confident, straightforward measures in the treatment of a condition that is usually considered to be much more unfavorable than it really is. Practically any one who takes the trouble can secure quite as good results.

The patient was a man, sixty-eight years of age, who never since he was a child had suffered from rheumatism or any of the infectious diseases that might cause infective endocarditis, and who in the meantime enjoyed excellent health—absolutely without any heart symptoms. His reason for coming was that he suffered from dyspnea so much that he required three pillows at night and was rather afraid to go to bed—partly because of the discomfort of his shortness of breath and partly because of his fear of possible death during the night. Before this dyspnea on lying down had been noted, he had noticed considerable difficulty in breathing whenever he attempted to go up-stairs even a little rapidly or in walking on a level even a moderate distance. He had been conscious of his heart's action for some months. Palpitation was quite marked, and this had evidently added a new element to his discomfort and emphasized his fear of serious consequences. He had no precordial pain and the discomfort had no tendency to radiate down the arm. Occasionally he suffered from vertigo. A marked feature in his symptoms was the presence of headache, which occurred whenever he became fatigued. The pain always affected the top of his head, was described as boring in character, and was evidently due to circulatory disturbance.

His general condition was excellent. His appetite was good, though he ate rather sparingly. His digestive functions accomplished their work without any disturbance, either subjective or objective. His bowels were regular, there was no eructation of gas, and none of his feeling of oppression was due to the dilatation of his stomach by gas and consequent pressure upon the heart

through the diaphragm. His vertigo could not be attributed to the presence of gas on his stomach, but was a purely circulatory disturbance, due to the fact that his heart's action became irregular after sudden movements or after exertion, as on going up and coming down stairs. In these cases, there is usually complaint of vertigo when the patient first sits up after lying down or when he raises his head after being stooped for some time, as in tying his shoes or other similar actions.

His physical signs were marked and not difficult to make out. His pulse was over 80; it intermitted occasionally, but was irregular also in its action, smaller and larger beats following one another without reason. His apex-beat was in the fifth interspace outside of the nipple line. This displacement would seem to indicate a serious condition, but as there was no displacement of the heart downward it was evident that the hypertrophy of the heart was at most slight, if indeed there was any of it. This form of enlargement affects the left ventricle and causes a displacement of the apex downward as well as to the left. A soft systolic murmur could be easily heard at the apex and was distinctly transmitted for some distance at the left, though it could not be heard at the angle of the scapula. The heart dulness extended beyond the sternum toward the right, showing that there was some dilatation of the heart.

There was further evidence of the dilatation of the right heart in the conditions observed in the lungs. Subcrepitant rales could be plainly heard over the bases of the lungs posteriorly, though the breath-sounds were quite normal over the rest of the chest. It was evident that there was congestion of the lungs consequent on the failure of the right heart to perform its work satisfactorily, and that this manifested itself, as is always the case, in the dependent portions of the lungs farthest away from the influence of the heart's action. The dilatation was mainly on the right side, and this accounted for the displacement of the apex-beat only outward and not downward. The enlargement of the right ventricle causes the right ventricle to occupy a position farther over in the chest toward the left than it did before, thus causing the displacement of the apex in this direction.

There was some edema of the legs, especially toward evening,

but the disturbance of the general circulation was otherwise not marked. The urine was of light amber color, perfectly clear, of acid reaction; it had a specific gravity of 1023, and contained no albumin and no sugar. For a man of his years and considering the rather spare diet that he took, the specific gravity was somewhat high. This was probably due to the fact that he was accustomed to take insufficient quantities of liquids generally and practically never drank water between meals. He was directed to take water in the morning, between meals, and before going to bed—with a manifest improvement of this single feature of the case.

After his shortness of breath and headache his most serious cause of complaint was a persistent cough, which occurred quite frequently, but without the satisfaction of expectoration, as a rule. Occasionally a little sputum was brought up in the morning, but in the day, though he had to cough frequently the cough was dry and irritating, causing him a great deal of annoyance. It seemed to him to be due to something in his throat, as he felt no disturbance within his chest, and accordingly he went to a throat specialist. In spite of the most careful treatment by all the most approved methods of applications and the douche and atomizer his cough did not improve. After an examination of his heart and lungs it became clear that his cough was due to the chronic congestion of his lungs consequent on the failure of the right heart to perform its function.

His case illustrates very well the necessity for realizing that local conditions in the throat and nose are often due, not to local causes, but to some systemic condition. Special treatment of the affected part by local measures may lessen the discomfort for a time or may palliate the condition, but it cannot really relieve it and will ultimately fail of effect entirely. Very few cases of beginning loss of compensation in the heart occur without this tendency to irritating cough, especially in elderly persons, so that in patients beyond middle life the presence of the dry cough should arouse suspicion of cardiac disturbance.

The patient's cough yielded rather promptly to small doses of morphin, one-eighth grain (0.008 gram) once or twice a day, which was given to him without letting him know what he was taking. Personally I often find it advisable not to let patients know when they are taking morphin. Some of them have a prejudice against

it and the consciousness that they are taking it is sometimes sufficient to hinder the action of the morphin and produce a restlessness that is not likely to be beneficial to any irritative conditions; besides if there is no knowledge that the drug is being taken there is little likelihood of the morphin habit developing. There is no doubt that morphin is one of the most valuable remedies for quieting excited hearts, especially in older patients, though of course the danger of the formation of the morphin habit must never be forgotten.

Because of the rather serious dyspnea and the displacement of the apex-beat it was evident that this heart first of all needed a rest. Rest is the most important thing in the treatment of a heart that is beginning to show signs of being unable to perform its functions. The difference between the amount of work the heart has to do with the patient moving around and lying in bed, the relief afforded the heart by not having to pump the blood against even the effect of gravity is quite sufficient to enable it in most cases to catch up with its work, if its failure has not been very marked. To confine a patient to bed, however, often proves so wearisome and makes him so restless as to undo to a great extent the beneficial effect of the recumbent position. I usually allow my patients then, unless the case is very severe, to get up and dress after being twelve hours in bed, provided they assume a reclining position most of the day. This patient slept on the third floor and was advised not to come down stairs more than once in the day, say for his principal meal in the evening, and to keep as quiet as possible the rest of the time.

Fresh air is an extremely important factor in the therapeutics of these cases, and patients very soon suffer from living in confined close quarters. The open-air treatment is quite as important for heart affections as for respiratory troubles. The patient was advised, therefore, to keep his windows open day and night and not to allow his room to become close and stuffy, even in damp weather or on cold days. These directions were intended for night as well as day, for the only pure air is the night air in spite of prejudices against it.

I have purposely left the drug treatment of the affection till the last, not because it is not important and extremely efficacious, but because, as a rule, it will not prove effective unless the other

details of treatment are carefully attended to and insisted upon in conjunction with the remedies. The heart is a very willing organ, which is readily imposed upon, and during loss of compensation it would be too bad to stimulate it into greater activity by drugs, if the patient does not take every precaution to save the heart every possible strain that can be avoided. In this matter it is especially important to remember that rest is very necessary. Rest with the patient lying down gives the heart its best chance to recuperate. This organ has at least one-sixth of its work spared it if the patient is recumbent over what it would have to perform if he were only to walk quietly about. If any further exertion is taken the heart-beats rise so rapidly that at least one-half the strain put upon it is saved by resting.

Of course, from the very beginning of the treatment the greatest care is necessary to secure reasonably free action of the bowels. There must be no gaseous distention of the intestines, since this forces the diaphragm up and thus mechanically interferes with the heart's action. A thorough evacuation of the bowels must be secured every day, and the patient was recommended to take sufficient Apenta or Carlsbad water every other day to secure this with assurance. At the beginning of the treatment a small amount of calomel is advised on several different occasions about three days apart, in order to secure the evacuant and diuretic effects of this drug. When the edema is not marked and when it yields to ordinary heart-tonics, the further use of calomel seems inadvisable. Of course, patients must be told not to eat such materials as ordinarily give rise to gaseous accumulation or to eructations.

In these cases of heart dilatation with rapid pulse, distinct subjective sensations, of palpitation, and a feeling of discomfort in the precordial region, I always find the indications for drug treatment best fulfilled by combining digitalis, strophanthus, and nux vomica. The digitalis stimulates the heart muscle, but slows its action and causes diastole to be longer than it would otherwise be. It is during the diastole that the heart gains the only rest that it has during the day. In the normal heart about two-fifths of a second of rest are thus afforded in every second of time. The lengthening of the diastole also affords opportunity for the better nutrition of the heart. Strophanthus acts almost like digitalis,

but seems to have a special effect in lessening the subjective symptoms of heart affections. The nux vomica is a general muscle-tonic, and acts not only upon the heart but also upon the arteries, and it must not be forgotten that these form a complementary dynamic element in the circulation. In general the contraction of muscles throughout the body is rendered better, and this aids the return of circulation and relieves the heart of some of its work.

My favorite prescription for these cases is:

℞ Tincture of digitalis,
Tincture of strophanthus,
Tincture of nux vomica, of each 2 drams, 8|0
Mix. S. Twenty-five drops in water, three times a day before meals.

Given thus before meals this prescription usually acts as an appetizer also, and these patients often have need of one. The dose may be increased, if considered necessary, up to 30 drops or even more, but this is usually quite sufficient if rest is insisted on.

Under the use of this remedy the edema gradually subsided, the cough improved, partly of course because of the morphin, and the dyspnea and subjective sense of discomfort in the heart region became much less marked. At the end of ten days the pulse dropped to 60 a minute and remained steadily there while the patient was lying down, so that it was considered advisable to take the digitalis out of the prescription. As a rule, it is important not to give digitalis longer than is absolutely necessary, that is, not after the pulse has come down to normal, since in many persons the drug is cumulative in its action and sometimes seriously disturbs the stomach and the general sense of well-being. The heart impulse improved in character and it was interesting to note that it gradually began to find its way within the nipple line. At the end of three weeks the rales had disappeared from the lungs, showing that the movement of the blood in the pulmonic circulation was sufficiently forceful to prevent the occurrence of congestion.

During the period of gradual disturbance of heart compensation the patient's general condition had suffered, so that as soon as he was rid of the more serious heart symptoms a general tonic was given.

The following combination has always proved especially effective

in such cases in my hands and I can recommend it for its tonic alterative effects:

℞	Arsenic trioxide	½ grain,	0|03
	Dried sulphate of iron	45 grains,	3|00
	Quinin sulphate	30 grains,	2|00
	Ipecaac	2 grains,	0|13

Mix and make capsules XXX.

Sig. One capsule three times a day after meals.

Owing to its irritant properties it is always better to give arsenic after meals and while the stomach still contains food.

As convalescence progressed and after the apex-beat had resumed its normal position, the patient was allowed to take short walks just up to the limit of fatigue, and these were gradually increased as his condition improved. In older people it is much easier to do harm than good by allowing any freedom in the matter of exercise. In younger people it is often very serviceable to increase the amount of exercise until quite long walks are taken, and if an opportunity is afforded for hill climbing to a moderate degree this is commonly of the greatest benefit. Nearly always in older people, however, such strenuous exercise is likely to prove eventually harmful. In persons over sixty years of age certain changes in the arteries have taken place and the system does not respond well to prolonged efforts that require exertion.

Nearly the same thing can be said with regard to Nauheim baths. They are excellent in persons under forty-five years of age, provided, of course, there are no signs of precocious arteriosclerosis, and when accompanied by the resisted exercises which were suggested and arranged by the Schott brothers, they bring about more rapid and more satisfactory relief of heart symptoms than any other method of treatment that we possess. The artificial Nauheim baths, when properly given at an institution in this country, can be depended upon to be almost as efficient as those obtained at the famous watering-place itself. There is, of course, the lack of the suggestive influence of the surroundings at Nauheim, for the fact that other patients are getting better serves to encourage and strengthen sufferers almost as much as any physical method of treatment.

In general, however, it may be said that these forms of acute

heart dilatation with distinct mitral systolic murmurs that are conducted around the thorax and with the displaced apex-beat that seem to make it clear that a serious heart lesion is present, may yet be so much improved by the rather simple methods of treatment that I have suggested, as to make this a very satisfactory chapter in applied therapeutics. Too much stress cannot be laid upon the fact that the prognosis of these cases, far from being hopeless, as it is often supposed to be, is quite commonly, at least in the first development of symptoms, so promising, as to make such patients extremely interesting and quite satisfying subjects for care and study.

PROFESSOR FOURNIER'S RECENT MODIFICATION OF HIS TREATMENT OF SYPHILIS

BY H. SAINGERY, M.D.

of Paris

Fournier's classical method of treating syphilis is so well known that it scarcely need be defined. It is called a "chronic, intermittent treatment," and consists, in short, in taking care of the patient systematically for the first four years of the disorder, and during this period, of putting him through a series of mercurial treatments, separated from each other by intervals of therapeutic rest,—and all this whether specific signs are present or not. Fournier has recently modified this course of action to a certain degree, in a manner and for reasons that will be apparent.

An energetic treatment of syphilis begun at the beginning of the cases, and continued during the first four years, is, to use Fournier's words, a method that has rendered good service and in its day marked a big step in advance. But he thinks it has its weak points and has failed in some instances; and this is why he has endeavored to modify it. Should it be modified by lengthening the duration of the mercurial treatment, which stood at two, three, or four years? He thinks this would be excessive, as well as useless, at any rate in the majority of cases.

Would it be preferable to substitute for the usual method what is called the *intensive* treatment? He thinks not; because the latter, though excellent and even indispensable as a temporary means, with a view to putting a rapid end to a menacing manifestation, would be most undesirable and the last thing in the world to recommend for the routine treatment of this disease. So what he now proposes is to allot in a different manner than is now done the series of mercurial "cures."

This modification Fournier has now been testing for several years, and he is inclined to believe,—for he does not yet feel able to come to definite conclusions,—that the old four-year treatment is

capable, without any lengthening, of giving better preventive results, if it is split up and the fragments are placed at wider intervals apart. The following, then, is the plan Fournier now proposes: At the beginning an energetic course, to be kept up methodically for the first two years, in the shape of a series of mercurial treatments alternating with intercalary periods of therapeutic rest. Then, stop all treatment for two years; and at about the fifth year, carry out another course, of one year's duration, as before. After that, interrupt again for two or three years, advising a third course of one year at about the seventh or eighth year of the disease.

Such is Fournier's present programme, open of course to amendments and changes according to individual cases.

In justifying this change, Fournier observes that except in unusual cases an ordinary mercurial treatment of two years' duration generally suffices to put an end to all manifestations of the disorder and to reduce them to silence for at least several years; it therefore seems superfluous to continue with the mercury without interruption. Since we are well ahead in the contest, let us take advantage of our advance and give the patient breathing space.

For mercury, like vaccination, is a preventive; but, again like the latter, its action is only temporary. Just as you must revaccinate, so also you must remercurialize, to protect the patient against further onsets of the disease. In the same way as mercury enables a woman recently infected and pregnant to give birth to a healthy child, so it ought to be capable of warding off any manifestation of syphilis. Everybody will admit that it is very unusual to see an explosion of tertiary symptoms take place soon after a mercurial "cure," especially a vigorous and prolonged "cure" following other similar "cures."

The worst forms of syphilis are not those that are remarkable by the variety of their manifestations and by their successive outbreaks; but those that are seemingly harmless at the start, and yield at once to treatment,—and then end, after a long period of silence, in some frightfully grave tertiary accident.

Fournier thinks that this difference in evolution is due to the fact that in the first instance, owing to the multiplicity and repetition of the manifestations of the disease, the patients had been necessarily treated over and over again and at various stages of the disor-

der, stages more or less widely apart and consequently remote from the beginning of the case; whereas in the second instance, in which there were no symptoms except the initial ones, the treatment was only prescribed during the early stage of the disease, which on this account has not had its virulence gradually attenuated by a series of mercurial "cures" and has retained, in consequence, in a latent state its degree of original virulence. A good case of syphilis is one that by means of frequent symptoms recalls itself to the patient's attention and forces him to take it into account and treat it; whereas a bad case is one that is lost sight of through lack of symptoms.

These facts being admitted, Fournier concludes by saying that the indication for courses of treatment of this sort, that is, remote from the beginning of the disorder, appears to be an evident one; and such a one is the treatment by successive and remote stages, which he now advises.

As regards the times at which these periods of mercury should be advised, Fournier says that this is a pure matter of experience. It is evident that we can only be guided by dates at which we have found tertiary manifestations most likely to appear; as the late courses of treatment must clearly be given at moments when there is a likelihood that the patient will have some outbreak.

In coming to a decision on this point he consulted his personal table of dates at which paresis manifested itself among his patients. This table, covering 112 cases, is as follows:

Period.	No. of Cases.
Toward the end of the third year	1
During the fourth year	3
During the sixth year	7
During the seventh year	8
During the eighth year	10
During the ninth year	10
During the tenth year	15
During the eleventh year	12
During the twelfth year	11
During the thirteenth year	5
During the fourteenth year	7
During the fifteenth year	6
During the sixteenth year	2
During the seventeenth year	2
During the eighteenth year	5

Period.	No. of Cases.
During the nineteenth year	3
During the twentieth year	2
During the twenty-third year	2
During the twenty-fourth year	1

This table then shows, first, that paresis begins to appear with startling frequency at the sixth year; and, second, that it is at its maximum between the sixth and the twelfth years, with an acme at the tenth.

He, therefore, argues that a serious mercurial therapeutic effort seems indicated, from a preventive standpoint, at about the fifth year, on the one hand, and at about the seventh or eighth, on the other; and that a course of treatment of this nature would seem to be more suitable at these stages than at any other. In any case is the indication for them not more rational at these periods than during the third and fourth years, as we have fallen into the habit of advising, through a certain amount of routine, no doubt? Such a course of action is in no wise inflexible, and must always be subject to modifications according to cases and farther experience in dealing with them; but Fournier feels that in general terms this therapeutic method is distinctly preferable to that of concentrating all our efforts on the first few years of the disease.

In the foregoing résumé of Professor Fournier's views on this subject a point to note is what he says against the use of the intensive treatment of syphilis in the treatment of the ordinary, every-day case. It is worth while to call attention to this opinion at a time when every one is speaking of hypodermic injections of mercurial salts in large doses!

This remark made, we are inclined to think that Fournier's modification of the ordinary treatment of syphilis is of no little importance. His revised method is somewhat similar to that which holds that syphilitic patients should be treated only when there are manifestations of the disorder or when such manifestations appear imminent,—so that it would seem possible that judicious therapeutists will some day find themselves in agreement!

For Fournier says: "Treat the disease methodically for the first two years, taking into account, naturally, the indications peculiar to each case." But it will be queer, indeed, if a syphilitic pa-

tient does not have symptoms during the first two years that will bring him to his physician many a time: various eruptions, mucous patches, headache, pains, more or less defective general condition,—for which he will be put under treatment. Besides, it is during the first two years that the patient is particularly menaced by syphilitic arteritis, a most insidious disorder, against which his physician will be desirous to protect him. So that the partisans, on the one hand, of the methodical mercurial treatment, who believe that mercury attenuates the virus; and on the other, the partisans of the method of treating syphilis only when there are manifestations, since they believe that the mercury has only a local curative action, will both, in practice, virtually treat their patients during the first two years of the infection.

Many physicians failed to understand why mercury and iodide should be systematically given for three or four years to men who seemed perfectly well. Nowadays Fournier admits that this method is not the most rational one, and that it has perhaps degenerated into a question of routine. In exchange for this admission we see no objection to recognising the value of a late course of treatment at the time when, about the fifth year, a syphilitic patient comes to ask permission to marry. The man seems cured, but perhaps he is not. A moderate course of treatment will do him no harm, and may on the other hand ensure perfect health in the children that may be born. So that to give this advice is not carrying precautions to extremes.

Is it advisable to go farther still, and to recommend other and still later courses of mercurial treatment? That of the eighth year does not seem to me indicated, on the score of possible paresis, since I am of those who do not believe in the syphilitic nature of general paralysis.

Again, a late course of treatment at the ninth year, on the pretext that toward the ninth or tenth year a certain number of syphilitic patients develop gummas, let us say, does not seem indicated either; such patients can always be put under treatment at the first symptoms of gummas and be preserved from any catastrophe.

The reason why these late courses of treatment are counselled is that their advocates believe in the vaccinating power of mercury and in its attenuation of the virulence of syphilis. This power of vac-

cination is far from being demonstrated, in my judgment, since there is many a case known in which, in spite of the most careful treatment, the specific lesions relapse incessantly. But even if it were demonstrated, I believe it to be only a point of secondary importance, the great truth, in this connection, to be borne in mind being, in my opinion, that the lesions produced by syphilis are noteworthy in their evolution, which is toward spontaneous retrogression; syphilis is a disease whose common and natural termination is complete and definite recovery.

"I possess," says Fournier, "the notes of four patients who underwent long and careful treatment for light cases of syphilis (three during four years and a half, and the other during three and a half years), who therefore were treated in a fashion that many physicians would consider fully sufficient, if they did not look on it as excessive,—but who nevertheless developed and died from paresis. Furthermore, these four men were young, of good constitution, free apparently from any hereditary disorder, and had been for years free from all specific symptoms. And yet general paralysis overtook them like lightning out of a clear sky, without warning and without having been induced by any appreciable cause."

Fournier then gives the history of one of these patients, adding that that of another one of them was practically identical:

"A young man had the misfortune to contract syphilis at the very moment of his engagement to a young lady. He made some excuse for postponing his marriage, came to consult me, and then began to treat himself with frenzy. He took quantities of mercury, all he could get down, which was no small matter, as he tolerated the drug in a remarkable manner. This went on for four years, with the exception of a few periods of rest, to which I had all the trouble in the world of getting his consent. He then married, and in the fifth and sixth years after his disease he had two healthy children, who are now fine, well-grown youths. But at about the tenth year of his syphilis paresis set in and defied, naturally, even intensive treatments by mercury and ended in the usual manner.

"In the same way I once saw a similar overdosage of mercury followed, in the ninth year of the infection, with a condition resembling tabes, which ended in general paralysis that proved slowly fatal."

The above are Fournier's own words, and no one will question his facts. The strict conclusion drawn from them will be irrefutable, like all conclusions properly deduced from positive facts: "With mercury, and even with a great deal of mercury, we are never sure of preventing general paralysis" (Fournier).

It will be seen that Fournier's verdict conveys the belief that by means of mercury general paralysis *can* be warded off; in fact, he expressly says so: "The sole protection of a syphilitic patient against general paralysis consists in the mercurial treatment methodically prescribed, and carried out for a long, very long period."

But this is a statement that no one will ever be able to demonstrate. For, because a patient has taken mercury methodically and for long periods, and has not developed general paralysis, can we claim that it was the mercury that protected him? Of course not; for it is materially impossible to show that if he had not treated himself with mercury he would have developed paresis.

Those who believe that syphilis is not the cause of paresis,—and I am of their number,—have no reason, even of a sentimental order, of prescribing it as a preventive.

Those who believe that paresis is of a syphilitic nature, that its basis is a syphilitic lesion, and endeavor to prevent its occurrence by a mercurial treatment, appear to act rationally, but in reality are guided by a sort of "impression," which is the following: Mercury enables a syphilitic mother to bear a living and healthy child; why then should it not prevent arteritis, gummas, and exostoses? Why should it not ward off diffuse meningo-encephalitis? (Fournier.)

There is no objection to their hoping that it will, though it is very possible that such hopes are vain, for the "impression" that gave rise to them can be opposed by three other "impressions" that seem to reduce it to nothing: (1) Many are the syphilitic patients that have been treated with mercury without its having protected them from manifold and relapsing tertiary symptoms. Why, therefore, should the candidates for paresis be more effectually protected than these? (2) Thousands of syphilitic patients, particularly in tropical countries, never take any mercury, and nevertheless escape paresis. Why then should the "only preventive" means against paresis he claimed to be mercury? (3) It has been demonstrated that

even a syphilitic patient requires, in order to develop paresis, to be of a peculiar constitution, gouty, and congestive; and it has been equally well shown that an intensive mercurial treatment is useless or harmful to a paretic patient when the disorder has disappeared. Why, then, should it not be equally useless or harmful *before* it has appeared? Why should we not be afraid of encouraging a natural predisposition toward it? These are three arguments against the use of mercury as a preventive of paresis, to be opposed to the argument in its favor referred to above.

In a word: (1) It is materially impossible to prove that a syphilitic patient who escapes paresis owes this to the mercury he has taken. (2) It has been absolutely demonstrated that any amount of mercury does not always preserve a patient from paresis. Consequently, the administration of mercury as a preventive of general paralysis in a syphilitic patient cannot be logically upheld; its use in this sense is not a direct and strict inference from facts, but is based on hypotheses dependent on the "impressions" caused by facts, impressions that are controverted by still other "impressions" and hypotheses.

Medicine

AUTOINTOXICATIONS AND THEIR TREATMENT

BY EDWARD C. HILL, M.D.

Professor of Chemistry in the Medical and Dental Departments of the University of Denver, Denver, Colorado

In the complex of chemical functions termed life, man is ever on the verge of disease, and the dangers that beset him from without are not so great as the perils which lurk within his own system. The poisons generated in the body, which lead to the abnormal cell-reactions of disease, are in general of intracellular (catabolic) or gastrointestinal origin, and are formed by autolysis, disassimilation, abnormal secretion, fermentation, and putrefaction. A general rule is that the waste products of any organism are deleterious to it and may cause death, sometimes suddenly, when reabsorbed in sufficient quantity. The toxicity of these products varies in proportion with the complexity of their molecules. By vitiation of the interstitial plasma they cause arteriosclerosis and degeneration of protoplasm, with increase of the nitrogen output. Autointoxication creates, by depraving nutrition, the " morbid opportunity " essential for the pathogenic action of the nearly omnipresent germs, which poison the body by means of their toxins. Conversely, autogenic poisons are augmented in infected organisms through increased febrile disassimilation and alimentary putrefaction.

Even these poisons have compensating features. Thus the products of fermentation promote peristalsis, bacteria aid in the digestion of cellulose, and urea stimulates excretion by the kidney. The varying susceptibility (idiosyncrasies) of different individuals to toxic substances depends probably on peculiar cell-reaction and especially on defective elimination. Autointoxication is, in my opinion, the chief underlying factor in most chronic pathogenic conditions, and in the acute form of food-poisoning is encountered

more frequently than any other syndrome. The subject of autotoxemia was considered only empirically until two or three decades ago. To the Frenchman, Bouchard, and his English translator and editor, Oliver, we owe a great debt for the scientific exposition of self-poisoning.

The causes of disease must usually be associated to force the barriers of self-protection. Mechanical factors are important in producing autotoxemia, which, conversely, tends to lower the motor function of the affected parts. Dilated stomach is both a cause and an effect of self-poisoning. Intestinal gases may produce dyspnea and suggest heart lesions and asthma (*asthma dyspepticum*). Asthmatic subjects are specially prone to attacks after indiscretions in diet, particularly if the patient has a dilated stomach and a weak heart. Constipation, by favoring decomposition of the intestinal contents and multiplication of bacteria, is a predisposing cause of appendicitis, enterocolitis, and follicular enteritis, especially in young persons. On the other hand, appendiceal adhesions lead to intestinal paresis, flatulence, and autotoxemia. In strangulated hernia and other forms of intestinal obstruction, depressing poisons are formed quickly in quantity. Abrams holds that congestion of the veins of the abdomen is a leading factor in chronic intestinal toxemia.

The nervous element is not to be disregarded in the development of autotoxemia. Reflex or trophic factors act mainly by inhibition of natural functions. Thus, cold, by vasomotor action, causes passive congestion of the alimentary tract, with stasis and decomposition of its contents; hence the rational use of laxatives in coryza. "Catching cold" is, reciprocally, more frequent under autotoxemic conditions. Depressing emotions check normal digestion and peristalsis and so favor fermentation and putrefaction. They also, it is claimed, excite directly the formation of toxic compounds, whose presence can be shown in the exhaled breath (Gates), and which account for poisonous effects in infants sucked by the milk of mothers undergoing violent psychic disturbances. The fear of cholera may bring about this disease by inhibiting the secretion of hydrochloric acid, which is destructive to the comma bacilli. Drug habits add greatly to the ill effects of autointoxication, which, on the other hand, increase the craving for the drug.

The matter of environment has much to do with self-poisoning.

When everything is not in its place, health is likewise disordered. Bile, peptones, and albumoses are poisonous in the blood, though not in the bowel. Pancreatic juice, let forth by trauma, causes fat necrosis in the surrounding tissues. Billions of bacteria (one-third the weight of fecal solids), mostly insusceptible of cultivation, inhabit the alimentary tract in health, but cause disease on passing into other parts. The colon bacillus, a normal habitant of the bowel, is a frequent exciter of peritonitis and of pyelitis, by invasion of the peritoneum and the renal pelvis.

The etiologic classification of Albu includes: (1) Gastrointestinal autointoxication, from abnormal decomposition of food substances; (2) arrest of organic function, as in Addison's disease, myxedema, cretinism, cachexia strumipriva, pancreatic diabetes, acute yellow atrophy; (3) anomalies of general metabolism, as in gout, oxaluria, diabetes; (4) overproduction of physiologic and pathologic products, as in overwork, cystinuria, diacetemia, ammonemia, etc.; and (5) retention of physiologic metabolic products, as in uremia, cholemia, eclampsia, asphyxia, extensive burns. The urine of fifty-two hours (urea of sixteen days) or the bile produced in eight hours will kill a man. The carbon dioxide exhaled by one man in twenty-four hours would poison him lethally many times if retained. The blood itself is normally toxic to one-tenth the fatal degree. The saliva of some men is nearly as poisonous as serpents' venom (Brunton). The contained pigments account for one-third the toxicity of urine and two-thirds that of bile.

The fermentation and putrefaction of foods in the stomach and bowels are due to the introduction of germs therewith, and, as in ordinary infection, there may be an incubation period of several hours or days. The appendicular region is particularly rich in germs (Brunner). Special foods in certain people excite autointoxication because they are not digested. Carbohydrates ferment into H, CH_4, CO_2, fatty and lactic acids, and other products not usually of a bad odor. Fats undergo fermentation and oxidation and become yellow and rancid, with liberation of fatty acids. Proteins putrefy, through the agency of anaërobic saprogenic bacteria, into bad-smelling alkaline products, particularly H_2S, benzene bodies (phenol, skatol, indol, cresol), ammonia, amins (methyl and ethyl amins not poisonous), amidoacids (leucin, tyrosin, etc.),

chinolins, acetone, methyl mercaptan, and, above all, ptomains, or putrefactive basic bodies.

Many ptomains differ from vegetable alkaloids in having their nitrogen arranged in an acyclic, or open chain, molecule. Though closely resembling different vegetable principles in physiologic actions and chemic reactions, they cannot usually be obtained in the crystalline form, and are temporary and transitory in nature. Oxygen-containing ptomains (amids) are betain, cholin, gadinin, muscarin, mytilotoxin, neurin, etc. Free from oxygen (amins) are amylamin, butylamin, cadaverin, methylamins, mydalein, neuridin, putrescin, saprin, and spermin. Poisonous ptomains are only in a small minority and include cadaverin and putrescin (meat), neurin and neuridin (meat), tyrotoxicon (milk, cream, cheese), mytilotoxin (oysters and mussels), muscarin (flesh, fish, fungi) and cholin (animal tissues generally), which oxidizes into muscarin and becomes neurin by dehydration.

Ethylidenediamin (Kemp) is a ptomain produced in dilated stomachs. Methylguanidin, formed in the small intestine, causes nervous irritability and convulsions, which are likewise produced by muscarin, putrescin, and neurin (dyspnea, unconsciousness). Depression and shock in intestinal obstruction are due chiefly to pentamethylenediamin, H_2S, and methyl mercaptan, formed in the stagnating contents of the bowel (Kukula). Ptomatropin, found in decomposing fish, sausage, game, and canned beef, produces symptoms much like belladonna poisoning, and may react to Vitali's test. Dilation of the pupil in lower animals (Vaughan) may be obtained by the application of extracts from putrid animal tissues. The production of indol depends largely on the activity of the colon bacillus, which ferments carbohydrates before acting on proteins. It causes (Kemp) headache, colic, diarrhea, neurasthenia, spasms, and cardiac and respiratory depression. Intestinal bacteria form phenol and cresol from tyrosin and break up lecithin into cholin.

During putrefaction some ptomains appear (cholin, first; toxic mydalein, about the seventh day) and later disappear, to be succeeded by others, the toxic ones being most abundant from the fifth to the thirtieth day. Non-alkaloidal (insoluble in alcohol) products are more active than the alkaloidal. Protein foods not perceptibly tainted may be very toxic, while those decidedly putrid

may be quite innocuous. Canned meats which splash on shaking are dangerous, from the contained gelatin-liquefying bacteria. Of all protein foods the most dangerous is cold-storage poultry, which is sometimes kept for a year or more in the undrawn state, and immersed in water before selling to render plump and coincidently spread the ptomains from the gut throughout the body of the fowl. Nearly all food preservatives used continually have a deleterious effect on digestion and irritate the kidneys. They do not prevent autolysis with formation of poisonous products. Cooking meat or fish may kill the bacteria but not destroy the toxins therein. Keeping protein food for a day or more after cooking or after opening the container, is the most frequent cause of ptomain poisoning. Germs and spores may gain access to food from sewer air.

Ichthyotoxins are present in salt herring and codfish and especially in canned fish. Mytilotoxismus, the most fatal form of food infection, is caused by eating crabs, prawns, lobsters, cockles, and mussels. Cooked clams and oysters may still be poisonous. Galactotoxins, or milk poisons, are saprophytes causing the summer diarrheas of artificially-fed infants. Disease of the udders may cause pseudodiphtheria or tuberculosis. Many epidemics of typhoid, foot and mouth disease, diphtheria, and scarlet fever have been associated with the milk supply. Toxicogenic germs, chiefly of the colon group, are present in nearly all cheese, the colors of which are due largely to fungi. Tyrotoxicon is a poison formed at times in cheese, ice-cream, frozen custards, and cream-puffs.

Meat or sausage poisoning (botulismus, kreotoxismus, allantiasis) is due to the anaërobic Bacillus botulinus or Proteus vulgaris, and occurs mostly in connection with raw or imperfectly cooked flesh, particularly of swine; the average incubation period is about eighteen hours. Putrid meat looks soft and smeary and has a grayish-green tint. The extract of 2.5 grams of putrefied meat is sufficient to kill a man (Bouchard).

All these food-poisons produce fever or subnormal temperature, dry mouth and throat, vomiting and purging (sometimes bloody), suppression of secretions, hoarseness or aphonia, dilated pupils, squint, ptosis, diplopia, and a tendency to collapse, the pulse being usually disproportionately rapid. Vertigo, dyspnea, delirium, and a scarlet rash are occasionally noted.

The toxins of Bacillus enteritidis (allied to the colon bacillus) resist boiling for several hours. Bacillus friedebergensis is found in putrid sausage. Bacillus breslaviensis has been obtained from the milk of cows suffering with febrile diarrhea. Bacillus pleuro-pneumoniæ caused 490 deaths in the Middlesborough epidemic, which depended on soaking salt pork in water and only slightly drying. The swine plague germ and the anthrax bacillus are also to be taken into account in the consideration of food-poisoning. Mussels extract typhotoxin and other poisons from surrounding waters. Canned and salt or partly-smoked meats may lead to scurvy, because of the toxic effects of contained ptomains on the blood-cells. Meat poisoning may also occur from eating the flesh of pyemic or septicemic animals. The symptoms in this event are like dysentery, typhoid, or cholera. The stools are greenish or brownish and very offensive. Endogenous poisoning (siguaterra) may be caused by certain fish and the blood of eels. Young veal gelatinizes in the intestine and offers a favorable pabulum for putrefactive germs.

Some vegetable foods are likely to be contaminated with alkaloidal and other poisons. Sprouting and sun-baked growing potatoes contain considerable poisonous solanin, resembling atropin in action. Muscarin is the chief active principle in poisonous mushrooms or toadstools, which contain also neurin and helvellic acid, enzymes, and toxalbumins, particularly phallin. Sitotoxismus is the term applied to poisoning by vegetable foods infected with molds and bacteria. Slimy bread is so changed by the potato bacillus. Ergotism arises from eating bread containing the fungus (Claviceps purpurea) of rye and darnel, the toxic chemical agents being cornutin and ergotinic and sphacelinic acids. Adulteration of meals with chick-peas may give rise to lathyrism, manifested by chilliness, pain in the loin and legs, lameness, girdle sensations, paresthesia, and gangrene. The epidemic maidismus, or pellagra, of southern Europe is due to saprophytes in fermented grain. The oriental beriberi may originate from damaged rice; actinomycosis, from eating raw barley or other cereals on which the ray-fungus grows. Cassedebat found in preserves many toxic alkaloids, several of which resisted boiling. A persistent penetrating odor was noted on warming with alkaline bicarbonates. Fermenting canned vegetables swell out the

ends of the containers. The fermentive acids formed excite inflammation of the gastrointestinal mucous membrane. The six kinds of fermentation in the alimentary tract comprise lactic, butyric, acetic, alcoholic, oxalic, and succinic.

Amylaceous indigestion (Caillé) causes pain and soreness in the bowels, constipation or diarrhea, acid urine, intercostal neuralgia, hemicrania, and vertigo; whereas protein putrefaction is marked by a bitter taste, malaise, fever (high in infants), nervous depression, sleeplessness, melancholia, headache, vertigo, tinnitus, visual disturbances, and in infants convulsions. The stools are sour and scalding in fermentation—alkaline and very offensive in putrefaction. The fatty acids of fermentation are eliminated largely by the skin, which they irritate; hence the reason for restricting carbohydrates in cutaneous affections. Rickets is thought to depend on systemic excess of lactic and other acids formed by gastrointestinal fermentation, from relative excess of carbohydrates and relative deficiency of available oxygen. Autointoxication by fats and proteins (as in mince pie) is particularly prone to affect the temper, changing temporarily a Dr. Jekyll to Mr. Hyde. Stercoremia (constipation—potassium, ammonium, alkaloids) leads to anemia, chlorosis (Andrew Clark), and purpura, and acutely to vomiting, small, rapid pulse, clammy sweat, and sometimes albuminuria, fever, and death from collapse. Physical signs of gastrointestinal poisoning (Abrams) are relaxed abdominal walls, gas accumulation, increased abdominal sensitiveness, and enlarged and tender liver. Chronic ulcerative stomatitis is another sign (Forchheimer). To poisons from the prima via, particularly with ectasia or ptoses, are attributed nodosities about the second phalangeal joints and pain and swelling about other small joints.

Leukomains are formed in the living body by retrograde metamorphosis with insufficient oxygen (anemias). They comprise the nucleinic purin bases (xanthin, hypoxanthin, guanin) and creatins, which are normally destroyed by oxidation. Migraine is directly due to xanthins (Rachford). Guanin, from cancer degeneration, causes coma. Xanthocreatin, the most toxic leukomain, is formed in the muscles and produces depression, vomiting, diarrhea, and somnolence. The symptoms of fatigue are ascribed to excess of waste products (lactic acid, neurin, xanthin, hypoxanthin) acting on the

nerve centers and on the nerve endings in muscle. Saburral fever (Abrams) resembles typhoid, with nausea, vomiting, chill, headache, palpitation, and tachycardia. It is noted after overwork and emotion and depends on self-poisoning. Lassitude after heavy meals may be due to absorption of toxic peptones which have been formed in excess. The symptom-complex of leukemia has been attributed to a chronic peptonemia. According to Klebs, cholera nostras is due to methylguanidin, a virulent poison derived from the harmless substance guanidin, by substitution of a methyl for a hydrogen atom.

Uric acid is normally destroyed in the human organism by oxidation into urea, chiefly in the muscles, kidneys, liver, spleen, and blood. Deficient oxidation is the underlying factor in goutiness, or the "uric acid diathesis." The endogenic (nucleinic) uric acid amounts to about one-fourth of the whole, but there are marked, though constant, individual differences (Long). The exogenic uric acid is derived from oxypurins (nucleins) and aminopurins (purin bases) in the food. An attack of arthritic gout occurs when the synovia is impregnated with uric acid to the extent of 1 to 2500 (Roberts). The tophi of gout are white, rounded, plaster-like masses of sodium biurate, with small amounts of lime salts, in the joints, tendons, and cartilages. The proportion of sodium salts is greatest in these fibrous tissues. Potassium salts prevent such precipitations. There is a close relation between uric acid and many cases of bronchial asthma. These attacks are sometimes accompanied by diarrhea and bilious vomiting, and are noteworthy for their periodicity (usually in the early morning).

The symptoms of cholemia are jaundice, slow pulse, pruritus, epistaxis, and rapid emaciation. The bile pigments are ten times as toxic as the bile salts. These salts dissolve and break up blood-corpuscles and other cells (setting free potassium and other mineral poisons), and the symptoms of cholemia abate as soon as the normal amount or more of bile salts reappear in the urine. The liver normally converts ammonium carbamate (formed in the tissues from ammonia and carbon dioxide) and leucin and tyrosin into urea. The symptoms of defective hepatic function (Neucki), such as somnolence, coma, and convulsions, are due in part to ammonium carbamate; ammonium salts are twice as toxic as those of potassium. Leucin and tyrosin appear in the urine in place of urea in acute

yellow atrophy. Cholesterin is probably an accumulation residue of the hydrolysis of carbohydrates by the epithelium of the gall-bladder and bile passages, and is also a product of nerve metabolism. It is converted into stercorin in the bowel. An excess constitutes waste material and forms gallstones, their production being favored by catarrhal conditions, stagnation of bile, and the presence of attenuated bacteria. Intestinal sand is due to lithogenic catarrh of the colon (mucous colic). Bronzed diabetes, or hemochromatosis plus glycosuria, is manifested by diabetic signs and symptoms, bronze pigmentation of the surface, and the deposit of brownish-black iron-containing (hemosiderin) and yellow non-ferruginous (hemofuscin) granules in the internal organs. It is due to unknown toxic causes which lead to breaking down of the blood cells and interstitial inflammation of the liver and pancreas. Floating right kidney is thought by Bouchard to be brought about by recurring congestion of the liver and resulting gradual displacement of the kidney.

In pregnant women the liver is sometimes large and tender, with pale stools, slight icterus, ascites, and choluria. The salivation, vomiting, insomnia, bronzing, pruritus, high blood tension, eclampsia, and other disorders of pregnancy are probably due to autotoxemia from overwork of the liver and kidneys, when there is also inherent weakness of these organs. The fetus may produce substances toxic to the mother. Convulsions may also arise from excessive coagulability of the blood, leading to multiple capillary thrombi. The urine is less toxic and the blood serum more so than in health, probably from intermediate products of metabolism, because of the failure of some organ to elaborate them into useful or harmless substances. In eclampsia (Williams) the total nitrogen is greatly diminished, while the ammonia coefficient is practically normal. In pernicious vomiting of pregnancy (acute yellow atrophy) the total nitrogen is about normal, but the ammonia coefficient is greatly increased—from 3 or 5 per cent. to as high as 46 per cent. of the total nitrogen.

Common symptoms of uremia are contracted pupils, headache, pruritus, amaurosis, shallow dyspnea (Cheyne-Stokes), vomiting, diarrhea, convulsions, coma, and hypothermia or hyperthermia. According to Bouchard, the toxicity of normal urine is due to seven different poisonous products of disassimilation, namely, urea (about

one-eighth of total toxicity), a diuretic; potassium (nearly one-half), a convulsant; coloring matter (three-tenths) and extractives (one-tenth or two-tenths), including a myotic, a sialagog, and a narcotic. The type of uremia, whether comatose or convulsive, varies with the relative proportion in the blood of the corresponding toxins. The toxicity of urine is almost nil in uremic attacks, the urea in the blood being increased at times to thirtyfold the normal. The freezing point of the blood serum is lowered to –0.61° to –0.67° C. in nephritis with uremia, owing to this molecular concentration. The diminished electric conductivity of blood serum (due to excess of proteins from retained nitrogen) may be the direct cause of uremic cortical symptoms (Stern). Deficient urea excretion is a more reliable danger signal than the presence or amount of albumin. Uremia is infrequent in patients with marked edema, since the cells, already saturated with chlorides that cause the edema, do not so readily fix the urinary poisons. Two c.c. of normal urine is sufficient (one urotoxy) to kill a kilogram of living matter. The urotoxic coefficient in health is 0.464 urotoxy per kilogram of weight. The urine of sleep, though more dense, is nearly always less toxic than the day urine. Muscular exercise in the open air makes urine less toxic, probably by enhanced oxidation. Urine is generally more toxic in disease, the convulsive elements (potassium and coloring matter) predominating in fevers, because of increased breaking down of the tissues. It is less toxic in mania, more so in melancholia.

The microbic poisons in the urine are alkaloidal in nature, in the blood, albuminoid; hence the kidneys probably exert an analytic action on toxalbumins. The toxicity of urine is due in part to absorbed toxins of the colon bacilli (Gilbert). Griffiths and Albu have isolated special toxins from the urine in a considerable variety of diseases. In gastrointestinal autotoxemia Bouchard found the urinary ptomains increased fiftyfold. According to Griffiths, a toxic base, propylglycocyamin (creatin, with propyl substituted for one H), is found in the urine of patients suffering from various affections.

In the systematic examination of urine one encounters important data as to autointoxication. Indicanuria means, practically, diminished hydrochloric acid and excessive protein putrefaction,

and is most marked in the two clinically-opposite conditions of obstruction of the small intestine and Asiatic cholera. Chronic skin diseases are commonly associated with indicanuria (Singer). Excess of calcium oxalate usually signifies deficient oxidation (oxalic acid being an intermediate product in the degradation of dextrose) and HCl formation, and defective digestion of fats and carbohydrates (flatulent and nervous dyspepsia). Urobilin is formed from bilirubin by bacterial reduction in intestinal putrefaction. Pyrocatechin is commonly found in the urine as a result of intestinal putrefaction, and is derived from the corresponding acid in plants; the urine darkens on standing. The fatty acids occur in the urine normally only in traces, but may be increased ten to twenty times, from intestinal fermentation in febrile and hepatic diseases, and the same is true of the ammonia excretion in acidoses. Acetone, diacetic acid, and oxybutyric acid are present in notable amounts in cancer, severe diabetes, sepsis, and other grave nutritive disturbances. Poisoning by these substances is manifested by vomiting, weak and rapid pulse, dyspnea, cyanosis, jactitation, convulsions, and coma. Albuminuria is frequently of gastrointestinal origin. The Bence-Jones disease is manifested by marked albuminuria, indicanuria, pain in the chest and back, and tumors often appearing on the shoulders, ribs, and arms. Cystinuria, always accompanied by diaminuria, occurs along with putrefactive processes in the intestine and appears to depend on a specific abnormality of metabolism.

The nervous system is prominently involved in autotoxemia from whatsoever cause. Vertigo is often of gastric or intestinal origin. Drowsiness is complained of in jaundice and in obesity. Coma is noted (often with labored, snoring breathing) in uremia, diabetes, many fevers, acute yellow atrophy, dilated stomach, alimentary cancer, cirrhosis of the liver, miliary tuberculosis, and severe anemias. Infantile convulsions may depend on coprostasis or acetonemia (von Jaksch). Tetany is sometimes produced by alkaloids generated in a dilated stomach; also by worms. Neurasthenia and epilepsy (cholin in blood) are aggravated by intestinal poisons. Robertson holds that general paresis is of enterotoxic origin, and Butler claims that locomotor ataxia is sometimes mimicked by self-poisoning. In recent years alienists have written

repeatedly on the relations of insanity and Bright's disease. Acute insanity has supervened upon obstinate constipation, uremia, or diabetes, and Bouchard cites cases of phthisical insanity coming on when excessive weakness prevented expulsion of the sputum. Some other miscellaneous autotoxic nervous symptoms are irritability, headache, syncope, insomnia, hypochondriasis, delirium, paresis of accommodation, muscular cramps, "dead fingers," transitory aphasia, polymyositis, and periodic myasthenia and paralysis.

Man is protected from autointoxication by the healthy action of his emunctories and by neutralization within the fluids and cells of the body. In this self-protection the liver is the chief organ of defense, oxidizing (by oxidases) and neutralizing poisons and excreting them in the bile. Indeed, the punster who said: "Is life worth living? That depends on the liver," uttered an epigram as well as a pun. The liver oxidizes indol, for instance, into indoxyl and conjugates this with sulphuric acid, forming indoxyl potassium sulphate, a substance less toxic than indol and more readily excreted through the kidneys. The bowels are oftener loose than constipated in autointoxication, through nature's efforts to effect a cure by means of a salutary vicarious diarrhea. Vomiting is less frequent as a natural relief. The mucous membrane lining the intestines is excretory as well as secretory in action. Bile salts and pigments are precipitated in the bowel and not reabsorbed to a great extent. The acid fermentation products of milk and carbohydrates suspend the development of the ordinary saprophytic microörganisms of the alimentary canal, whereas meat favors their growth. The kidneys are the chief organs of elimination, and extra work is thrown upon them when the liver is diseased. The skin and intestines can pass out in the same quantity of water only $^1/_{500}$ as much urea as the kidneys (Bouchard), hence their vicarious eliminative action is not important. Indeed, Bouchard asserts that perspiration is not beneficial in uremia, since it lessens the amount of urine, the chief depurant. He adds that the abstraction of 32 grams of blood carries off more poisons than 100 liters of perspiration. Connective tissue absorbs bile pigment, and so protects brain and nerves. The lymph-glands serve as filters to bacterial products. Leukocytosis is the rule in threatened autointoxication.

An acid reaction annuls the irritability of bioplasm. The blood and most secretions are alkaline—less so in fevers, because of fermentation and excessive destruction of body proteins (sulphuric and phosphoric acids). There is increased elimination (up to 10 grams daily) of ammonia in acidosic conditions (fevers, cachexias), the catabolic ammonia of the cells, which would otherwise be burned to urea, combining with the acids to protect the fixed bases of the organism.

The chlorides of the body (Hall) preside over the chemical regulation of the tissues and fluids and prevent (detoxication) accumulation of toxic substances. Whenever poisons threaten the security of the economy, chlorides are despatched thither to increase the molecular tensions of the region affected. Decrease of chlorides in the foods causes the retained chlorides of the tissues to release their hold upon them and to be eliminated in conjunction with these toxic substances.

Old cultures of microbes contain substances capable of arresting the growth of these bacteria, and it is possible that immunity depends on bacterial autointoxication, that is, poisoning of the germs by their own products. The alexin in blood serum, which destroys pathogenic bacteria, may be an enzyme (destroyed at 56° C.), and is probably derived from the leukocytes. Horror autotoxicus is the term applied by Ehrlich to the inherent cellular power which guards the organism against autolysis, that is, the solution and digestion by its own fluids which takes place after death.

One function of the ductless glands appears to be the neutralization of intrabiontic endogenous poisons. The suprarenals (Zucco) protect the economy against muscular poisons, that is, wornout pigments. According to Blum, the thyroid seizes and destroys enterotoxins developed by the action of bacteria in the intestinal canal. When the thyroid is absent or diseased these toxins produce changes resembling myxedema, as edema, debility, stunted growth and dulled mentality (cretinism), tetany, and subnormal temperature. The general mucoid infiltration of myxedema is due to degenerative changes in the fibrous and connective tissues. Notkin states that the thyroid contains thyroproteid (an excretory colloid toxin, producing symptoms like cachexia strumipriva) and thyroiodin (globulin and enzyme), and he holds that the thyroproteid is normally

neutralized by thyroiodin. The parathyroids are said to be even more important than the thyroid, antidoting gastrointestinal poisons which excite dyspnea, tachycardia, tetany, convulsions, and general tremors. Exophthalmic goiter may thus have its genesis in parathyroid insufficiency. Atrophy of the thymus gland is characteristic of infantile marasmus. The pituitary body is involved in acromegaly. Pathologic adiposity results after the removal of or morbid changes in the sexual glands; and castration retards osteomalacia and cancer of the breast. Febrile processes, says Oliver, are due largely to operations taking place in the parenchyma of the spleen, through the intensity of phagocytic action. Splenic enlargement is proportional to the number of red corpuscles destroyed and therein harbored, exciting hyperemia and hyperplasia. Interesting interrelations are noted with the ductless glands. Thus, the pituitary is enlarged after thyroidectomy; the thyroid becomes larger during pregnancy and often at menstruation; the testes are usually atrophied in cretins.

In the treatment of autointoxications elimination ranks first, and the necessity of a daily movement of the bowels has been recognized from the earliest times. A full dose of calomel, followed by some saline, is most effective in self-poisoning from the stomach and bowels, though in children who have eaten not wisely but too well castor oil may be preferable. Constipation can often be controlled by drinking six to eight times daily a glass of water containing enough table salt to make the liquid half isomeric with blood serum. Stomach and bowel lavage is beneficial in almost any kind of poisoning. Apomorphin hypodermically is a quickly acting and efficient emetic. The skin should be kept active by washing, friction and sweat baths, followed by cold douches, or sprays. Diuresis is best induced by enteroclysis with four to eight liters of physiologic salt solution. Saline infusions are especially serviceable to flush poisons out of the system. Stern advises using a hyperisotonic (1 per cent.) solution of sodium chloride intravenously— up to 500 c.c. at 115° F.—every four to six hours if need be. Bleeding and vasodilators (aconite, nitroglycerin, veratrum) are indicated for the high tension pulse of eclampsia or anuria.

The liver can be stimulated (Abrams) by deep inspirations with pressure of the patient's fingers up under the ribs, and by trans-

mitted percussion, using blows of the fist on the outstretched hand. Abdominal massage and exercises for the belly-muscles are generally useful, or one may try the sinusoidal current in full strength to the abdomen, for venous congestion here. Abdominal support is needed for pendulous abdomen and enteroptosis; Abrams recommends a soft leather belt 2½ or 3 inches wide, buckled around the umbilical region.

The best gastric antiseptic is the natural one, hydrochloric acid, which prevents hypochlorhydric fermentation. Intestinal antisepsis can, of course, be only partially achieved, but "A half loaf is better than none." Intestinal antiseptics may do good in typhoid fever by preventing non-specific saprophytic autointoxication. Oliver claims that the phenols exert a depoisoning (combine with toxins) rather than germicidal effect after absorption. Bouchard prefers insoluble and non-absorbable antiseptics, such as naphthalin, bismuth salicylate, and iodoform. Other agents of more or less value are thymol, salol, beta-naphtol, oil of cinnamon (Caiger—3 to 5 drops, 0.18 to 0.30 c.c., three or four times a day), formic acid (Stern—2 to 5 drops, 0.12 to 0.30 c.c., well diluted with glycerin, every hour), pure brewer's yeast (1 or 2 teaspoonfuls of dried; 20 to 40 c.c. of liquid), organic peroxides, petroleum or olive oil, fruit acids (Kellogg), bismuth subnitrate, salicylates, sulphites, and copper salts. Charcoal taken internally disinfects by fixing and oxidizing alkaloids, pigments, and other poisons in the alimentary tract. I have found phenolsulphonates (sulphocarbolates) most efficacious, and it has long been my custom to give the sodium salt in full doses in acute alimentary poisoning, and in all chronic diseases accompanied by foul stools and indicanuria.

Alkali therapy protects the fixed bases of the cells from combining with pathogenic acids (diacetic and oxybutyric in particular) in the circulation. Von Noorden gives 15 grams or more daily of sodium bicarbonate or citrate in every case of diabetes showing the ferric chloride reaction. I am accustomed in these cases to administer a level teaspoonful of potassium bicarbonate in water with a little lemon-juice night and morning. In diabetic coma Naunyn injects intravenously 35 or 40 grams of sodium carbonate, repeating if need be.

In acute autointoxication stimulants are often required to pre-

serve the patient while the poisons are being eliminated. Atropin is very useful in antagonizing the neurin, cholin, and muscarin of meat poisoning. Inhalations of oxygen are of special service when the blood is disorganized and dyspnea is prominent. Abrams recommends as invaluable digitalin and strophanthin, alone or combined, for the relief of abdominal venous stasis. Motor functions are aided by strychnin.

"An ounce of prevention is worth a pound of cure," never more so than in autointoxications. A proper diet is of the greatest importance. Generally speaking, the amount of food should be lessened. Surgeons obviate autotoxemia preparatory to operation by starvation (Ochsner method). Milk, buttermilk, starches, and cereals prevent putrefaction in the bowels. Carbohydrates tend to lessen the amount of acetone bodies in diabetes, since they contain much oxygen. One should avoid excess of proteins (kidney and liver diseases), carbohydrates (adiposity, diabetes), fats (torpid liver, diarrhea, skin diseases), and nuclein foods (goutiness). We should observe idiosyncrasies to certain foods (eggs, strawberries, shellfish) as well as to medicines. Haig recommends in "uric acid" cases with high blood-tension a diet of bread and fruit with as little fluid as possible. Grawitz reports cases of pernicious anemia (intestinal autointoxication) cured with a diet of vegetables and cereals mashed or in porridge, with abundance of lemonade.

Lack of drinking water leads to constipation, malassimilation, melancholia, and many obscure aches and pains. Impure water may excite dyspepsia, diarrhea, dysentery, typhoid or yellow fever, cholera, and entozoal diseases. According to Jackson, free water drinking is a potent preventive of cataract.

Produce and meats are kept best at a temperature just above freezing point. After freezing, meat, fish, and milk lose flavor and spoil more rapidly when exposed to heat. Various bacteria are able to withstand temperatures as low as 0° C. or as high as 72° C. Sterilized foods are likely to cause scurvy by the breaking down into less assimilable compounds of the organic combinations of potassium and other metals. Inspectors should condemn discolored, putrid, or suppurating meat; also (Bulstrode) beef from cattle dead of anthrax or cattle plague, mutton from sheep with smallpox or

splenic apoplexy, and pork from pigs with carbuncle, scarlet fever, hog cholera, or typhus.

Finally, hygienic principles must be inculcated and personally practiced, for unless causes are removed effects are certain to continue. The family physician should be above all else a teacher of hygiene and sanitation, able and desirous of directing his patients into the sane and simple ways that lead to health.

MULTIPLE ULCERS OF THE PYLORUS AND GASTROSUCCORRHEA (REICHMANN'S DISEASE) WITHOUT STAGNATION IN THE STOMACH

BY MAURICE SOUPAULT, M.D.

Physician to the Paris Hospitals, Paris, France

The nature of gastrosuccorrhea and the relation of this affection to round ulcer of the stomach are as yet matters for discussion. The following cases and the conclusions which they justify are, therefore, of interest, especially as successful pylorectomy was performed in both and allowed exact examination of the lesions under consideration.

Case I.—Mrs. D., 35 years old, was admitted into the Beaujon Hospital in April, 1900. Eight years before this time she had undergone a troublesome pregnancy, suffering from obstinate vomiting. Following delivery there were fever and symptoms of infection. A month later phlegmonous angina and uterine hemorrhages occurred and persisted for over a month; at the same time the first evidence of gastric pain was observed.

Prior to this time there were no symptoms whatever referable to the stomach. The pains were noticed at intervals; they occurred 3 or 4 hours after luncheon and were not accompanied by regurgitation or vomiting, as a rule. The attack lasted for 4 or 5 days, though sometimes the pain was followed by the vomiting of small quantities of a bitter or acid liquid and immediately terminated. These attacks continued during the next 3 years, lasting for from 5 to 20 days. After this time the pain was of greater intensity and longer in duration; occasionally vomiting occurred. The attacks also came on not only after luncheon, but also after dinner, at 4 P. M. and at 11 P.M. This aggravation of the pain persisted during the next 3 months, at which time her physician diagnosed some uterine malposition and ordered a bandage. All symptoms then disappeared, to recur at the end of six months, the attack lasting a month. This periodicity of attack continued to the time I first saw

her, in April, 1900. I found her nervous and fearful of another attack. I elicited the fact that nervous influences, and especially any little inconveniences or worry, would greatly aggravate the pain. I prescribed a dietetic regimen consisting of milk, milk soups, and mashed vegetables, and ordered some alkalines in capsule and cold compresses over the stomach.

Under this treatment she rapidly improved and in a month had gained five pounds. I then lost sight of her until March, 1902, when she again consulted me, and stated that she had felt well for five or six months, after which time the pain had recurred. The attack would begin in the afternoon and last for two or three hours, when she would vomit a clear, acid liquid containing no remnants of food, after which relief was obtained until late at night, when she would be awakened from sleep by the pain and again obtain relief by vomiting. These attacks lasted for several days, and were followed at first by a period of relief, but during the past six months they have been experienced nearly every day. The pain has become more intense and radiates to the back, the vomiting is more profuse and lately has contained some remnants of food, but never any blood. The attacks always occur in the afternoon; during the morning hours she feels quite comfortable. Constipation is marked, the general condition is poor, and she has lost forty-four pounds in weight since the beginning of the ailment.

Upon physical examination, the stomach when empty is found painful to pressure, and upon palpation, which can easily be done by reason of the lax abdominal wall, one hears an indistinct gurgling sound. After insufflation with air the lesser curvature is found at 7 or 8 cm. below the xiphoid cartilage, while the greater curvature is 4 or 5 fingers' breadth below the umbilicus. The pylorus is globular, the fundus normal in shape, but the organ seems to be bilocular and *tapotement* elicits visible peristalsis. She was then given a rich meal including a quarter pound of currants and six plums, and on the following day the stomach was found to contain 75 c.c. of a turbid, greenish fluid containing mucus but no food, even after standing in the testing glass. It was examined and gave the following reactions: Hydrochloric acid was present; organic

acids were absent; no amylaceous or sugary substances were present; the biuret reaction was slightly positive. Quantitatively:

Total acidity	3.23 per cent.
Total salts	4.92 per cent.
Fixed salts	1.94 per cent.
Hydrochloric acid	2.98 per cent.

This analysis indicated, therefore, hypersecretion by an empty stomach and digestive hypersecretion with marked hyperchlorhydria. In view of the failure of medical treatment, the severity and chronicity of the attacks, and the results of previous cases, surgical intervention was advised and accepted by the patient.

Operation (pylorectomy) was performed, in March, 1902, at the Lariboisière Hospital by Dr. Hartmann, to whom I am indebted for the following description: From the anterior surface the pylorus was observed to be narrowed as though the seat of a stricture produced by tying a cord around the pylorus, but there were no adhesions and the pylorus was freely movable. Upon palpation the pyloric portion of the stomach was found greatly thickened, the glands along the lesser curvature were not enlarged, but those behind and below the pylorus were the size of a large pea. Upon opening the stomach along the median line an ulcer was observed at the level of the upper margin of the pylorus, the size of a very large lentil, with serrated margins. It had perforated the coats of the stomach and the floor was formed by the fat of the mesocolon. The mucous membrane of the pyloric portion was the seat of multiple ulcers, 3 or 4 of which were distinctly of the " perforating " variety and 16 or 17 were erosions. They varied in size between that of a lentil and that of a bean and were round or oval, close to one another, but not confluent. The mucous membrane was edematous and the ulcer-bearing area merged indistinctly with the normal portion; the walls of the stomach were smooth and no cicatricial tissue capable of producing stenosis was observed. This was confirmed by microscopic examination, and it was also noted that the erosions before mentioned were quite superficial.

Four or five days after operation the patient could take nourishment and in three weeks she left the hospital. On May 24, 1902, two months after operation, I saw the patient again and noted that

her appetite was good; there had been no attacks, no nervousness, no weakness; and she had gained 13 pounds in weight. Upon sounding the empty stomach a very small quantity of slightly greenish liquid was obtained which scarcely reddened litmus paper and did not give the usual qualitative reactions of gastric juice. After a test meal a small quantity of fluid was obtained which revealed the following:

Total acidity	0.45 per cent.
Total salts	2.08 per cent.
Fixed salts	1.15 per cent.
Hydrochloric acid	0.93 per cent.

There was no longer a gastrosuccorrhea on an empty stomach, no digestive hypersecretion, and the remarkable hyperchlorhydria which had been present before operation had changed to a hypochlorhydria. I saw the patient again on July 24, and noted a further gain in weight of 10 pounds.

Case II.—B, a widow, gave a history of stomach trouble of five years' duration, variable in intensity, and irregular in onset—the further description of the attacks being very vague. During the first three years there were attacks of nausea, vomiting of sour material during the daytime, but no pain, followed of late by cramp-like pains occurring late in the afternoon, irregular, and intensified by cold or fatigue. Nausea was experienced before the meals and if followed by the vomiting of a sour fluid she was able to enjoy the meal, although sometimes a feeling of weight would be experienced a few hours after the meal—necessitating vomiting. Under a dietetic regimen and alkalies she improved markedly, but on January 15, 1901, she had hematemesis and melena lasting for two days and weakening her very much. The symptoms continued irregularly until December, 1901, when chocolate colored vomiting was noted. The stomach was then sounded by Dr. Liseau after a 14 hour fast and 120 c.c. of a white, viscid, odorless fluid removed. It contained no food particles in suspension and gave the following analysis:

Total acidity	2.08 per cent.
Total salts	3.74 per cent.
Fixed salts	1.20 per cent.
Free HCl.	0.87 per cent.
Combined HCl.	1.67 per cent.
Total HCl.	2.54 per cent.

A test meal was given and revealed a digestion hypersecretion, 250 c.c. being removed with the following analysis:

Total acidity	3.69 per cent.
Total salts	5.62 per cent.
Fixed salts	1.38 per cent.
Hydrochloric acid	4.24 per cent.
Free HCl.	2.70 per cent.

A remarkable hyperchlorhydria is thus observed and the patient was referred for operation to Dr. Hartmann, to whom I am indebted for the following notes: Upon palpation the pylorus was felt to be greatly thickened, not as a tumor mass but rather in the form of a diffuse infiltration extending toward the lesser curvature. There were no adhesions and the glands on the greater curvature were felt to be the size of peas. The anterior wall of the stomach was then opened and upon exploration the pyloric portion of the stomach was found greatly thickened, with an ulcer about 2 cm. in width encircling the pyloric ring nearly completely. This ulcer had serrated margins and was separated from the duodenal mucous membrane by a mere fold or ridge, 5 mm. in thickness, and was complete, as has been stated, except for a distance of 1 cm. on the antero-superior wall.

While the ulcer had the characteristics of the acute perforating type, yet it had probably developed upon the site of a lesion of older date, the surrounding tissue being hard, sclerotic, and contractile, producing a certain degree of stenosis of the orifice. The mucous membrane was edematous and vascular, the muscular layer hypertrophied over the whole region of the pyloric portion, and three smaller ulcers were observed, step-like in arrangement, along the lesser curvature. Pylorectomy was performed and the patient made a normal recovery from the operation.

In November, 1902, the patient was feeling comfortable, without attacks, and she had gained several pounds in weight.

The stomach was sounded after a 12 hour fast and found to be empty, with no evidence of stagnation after lavage. A test meal was then given, consisting of 60 grams of bread and 250 c.c. of distilled water, and the contents gave the following analysis:

Total acidity	0.96 per cent.
Total salts	2.20 per cent.
Fixed salts	1.06 per cent.
Hydrochloric acid	1.14 per cent.

By comparing this finding with the gastric analysis made before operation it will be seen that the marked hyperchlorhydria was followed by hypochlorhydria and that the hypersecretion by the empty stomach had disappeared.

As is seen in these two cases, gastrosuccorrhea may be associated with ulcer and clinically can be divided into two types. In the first, the stomach contents is large in amount and contains food remnants, either in suspension or floating in the liquid. In the second type the gastric fluid is clear and without food residue, even after centrifugating and examining with the microscope.

Investigators of the pathology of diseases of the stomach apparently agree that the gastrosuccorrhea of the first type is caused and continued by a cicatricial ulcer of the pyloric region, but they are more diverse in their views as to the etiology of the second type. The majority of observers hold that this type is an autonomous disease chiefly characterized by a disturbance of the gastric secretion, and due either to some simple functional trouble of nervous origin or to the result of special lesions of the glands in the gastric mucous membrane. The presence of ulceration in this type is considered rare and rather as an incidental complication in the later stage than as a cause *sui generis*. The reason for this opinion lies in the latency of the ulceration and in the absence of trustworthy signs which might lead to a diagnosis. Hayem, on the contrary, believes that the ulcer is the only cause of the continual hypersecretion and I, myself, can unqualifiedly endorse this view, not only because of clinical observation but also from 12 cases of gastrosuccorrhea of the second type which were operated upon. The two cases which are described in this paper bear out this statement and afforded a microscopic study rarely obtained, because Reichmann's disease in the early stages is frequently mistaken for gastralgia or considered as a curative disease.

How is the influence of gastric ulcer upon gastrosuccorrhea without stagnation, to be explained? It has been stated that most observers explain this relation by the occurrence of cicatricial stenosis of the pylorus and gastrectasis, but this only explains the gastrosuccorrhea of the first type. If we compare the two cases herein reported it will be observed that while Case II supports the stenosis theory, in Case I no stenosis was present, yet hypersecretion of an empty stom-

ach occurred under the same conditions. I am of the opinion that the stenosis plays but a small part in the etiology of these cases, the ulcer affording the cause from irritation and producing a reflex which leads to muscular contraction and to hypersecretion. This phenomenon may be compared to the lachrymation and contraction of the eyelids produced by corneal ulceration or disease of the lids. The passage of food over the ulcer-bearing area and a certain irritability of the nervous system cause vasomotor and secretory phenomena leading to hypersecretion of the gastric juice.

The evidence of the intensity of the vasomotor disturbances was noted by Dr. Hartmann when performing the pylorectomy in Case I. The entire pyloric region was reddened, swollen, and puffy, as if the coats were infiltrated by edema, and in the walls there were dilated blood vessels causing considerable hemorrhage when performing the excision. It is possible if not probable that this congestion was due to the repeated spasmodic contractions of the pylorus during the height of the attacks.

In discussing the treatment of gastrosuccorrhea I am led by the results of the pathologic examination of the stomach in cases of simple gastrosuccorrhea to state that surgical intervention is always justified after medical treatment has failed or has given only temporary relief.

I wish to emphasize this point, because while at the present time those cases of gastrosuccorrhea known to be caused by pyloric stenosis, and marked by gastrectasia and the retention of food are readily sent to the surgeon, the cases of simple gastrosuccorrhea are treated solely by medical means. If the cause of this affection is believed to be some ulceration at the pylorus it is evident that remedies calculated to diminish the irritability of the gastric mucous membrane will fail in their purpose. From my own clinical observation I am able to affirm that the results obtained by surgical intervention in the cases of simple gastrosuccorrhea are just as brilliant and as encouraging as are those in the form with stenosis and stagnation.

I have had about a dozen patients of the simple type operated upon, all of whom recovered and are observed from time to time in excellent health. They have gained in weight and strength, are free from attacks, and the cure seems to be permanent—allowing them to live an active life although under some dietetic regulation.

As to the particular operation, gastroenterostomy is the one usually selected, although pylorectomy was performed in the cases herein reported. Functionally, the results are about the same, but after pylorectomy the gastric acidity is more likely to decrease and the gastric secretion to diminish, the stomach being thereby placed more at rest. These latter facts are also corroborative of the theory which attributes the gastric hypersecretion to vasomotor disturbances from the irritable and inflamed ulcer.

SOME ACCIDENTAL RASHES OCCURRING IN THE COURSE OF TWO HUNDRED AND FIFTY CONSECUTIVE CASES OF TYPHOID FEVER[1]

BY D. J. MILTON MILLER, M.D.

Visiting Physician to the Episcopal Hospital, Philadelphia

In addition to the specific rose-spots and their modifications (accuminate papules, petechiæ, etc.,) the following rashes are occasionally met in the course of typhoid fever: (1) Erythematous rashes; (2) sudaminal and miliary rashes; (3) the taches bleuâtres; (4) herpes; (5) urticaria; and (6) boils and pustules.

During my recently-completed four months' service in the women's and children's wards of the Episcopal Hospital, in which 250 cases of typhoid fever were under my care (175 adult women and 75 children), the cutaneous manifestations were more than ordinarily numerous (16 in 250 cases).[2] In a long and rich experience with enteric fever, I can recall many instances of accidental rash; but never, in one term of service, have I encountered so many as have come under my service this year. Among 350 cases of the disease admitted into the male wards during the same period, only 5 accidental rashes were noted,—that is, 2 of erythema, 2 of herpes, and 1 of urticaria. Although this difference in relation to sex may have been only coincidental, and although the meagre reports of adventitious rashes in typhoid fever show no preponderance of female cases, yet I am inclined to believe that these eruptions are slightly more common in this sex—especially those forms in which nervous influence is generally accounted an etiologic factor.

Of the 16 accidental typhoid rashes forming the basis of the paper, there were 10 instances of erythema (including under

[1] Read before the Section on General Medicine of the College of Physicians of Philadelphia, April 9, 1906.

[2] There were actually fifteen rashes among the 250 cases; the sixteenth case occurred in the male wards (Dr. A. A. Stevens'), and is included because it represents so typically the scarlatinal form of rash.

that term two of morbilliform character), 4 of herpes, and 2 of urticaria. The sudaminal and miliary rashes are not considered,—not because none was observed, but because their comparative frequency in typhoid fever renders them unworthy of special comment. The same may be said of acute bed-sores, of which two instances were observed in the shape of extensive sloughs fully six inches in diameter about the sacrum. Neither are boils and pustules referred to, because none occurred.

The erythematous rashes, being the most common and important, may be considered first. Of these, as already mentioned, there were 10; but if the morbilliform cases are excluded, only 8 (or 3.2 per cent.) were seen. In the Johns Hopkins series of 829 cases of typhoid fever, 15 of the adventitious rashes were of this variety, or 1.8 per cent. This form of rash, as it occurred in my cases, may be conveniently divided into simple, scarlatinal, and morbilliform erythema.

Simple Erythema.—Of the first group, or simple erythema, 4 cases occurred:

Case I.—Female; age 20 years; fever of severe type, temperature ranging from 103 to 104° F. On twenty-first day, while the temperature was 100.4° F., a diffuse, erythematous blush appeared over back and trunk; rash not punctiform in character, of a bright red color, and of the nature of a uniform blush; lasted two days; not followed by desquamation. The appearance of the rash was not attended by any alteration in temperature or in the general symptoms; no sore throat or strawberry tongue; history of scarlet fever and measles.

Case II.—Female; aged 14 years; mild attack; temperature reached normal on thirty-seventh day; on thirtieth day, when the temperature was 100.4° F., a diffuse, erythematous blush appeared on chest and abdomen; bright red in color, but not punctated; rash disappeared in twenty-four hours; no desquamation; no sore throat; no alteration of temperature attended rash; history of measles, but not of scarlet fever.

Case III.—Female; aged 10 years; mild attack; temperature reaching normal on twenty-third day; on twelfth day (two days after admission), a faint erythematous rash on chest and back; not scarlatiniform; no implication of the throat; rash lasted two

days; no desquamation; temperature at appearance of erythema 101.2° to 102° F., and not affected thereby; history of measles, but not of scarlet fever.

Case IV.—Male; aged 11 years; attack mild; faint erythematous rash over face, chest, and abdomen on seventh day; lasted six days; not followed by desquamation; no change in temperature, which had been 103° F. at outbreak of rash; no throat involvement; history of measles, but not of scarlet fever.

In two of the cases, both in children under ten, a boy and a girl, the patients exhibited autographic skins,—that is, on stroking the skin, a well-defined raised welt or wheal appeared. With the subsidence of the rash, this feature also vanished, thus showing the sensitive state of the skin, and, perhaps, the existence of a nervous element in the origin of the efflorescence. In all the cases of this group, the rash consisted of a bright red, uniform blush, and was never punctated. It appeared on the back, chest, and trunk, lasted from one to six days, and was never followed by desquamation or attended by general symptoms. In one, only, was the course of the fever severe. One patient had had scarlet fever, and three had not. All the patients were under twenty years. Three were females, and one was a male. The rash appeared on the first day in one case; and on the twenty-first, twelfth, and thirtieth days, respectively, in the other three cases. The majority, therefore, occurred late in the disease.

Scarlatiniform Erythema.—There were 4 instances of scarlatiniform rash:

Case V.—Male; aged 26 years; severe type of infection; high fever; patient delirious. On the twenty-second day, dark-red rash, consisting of minute papules and points, appeared on chest, abdomen, and back, rapidly covering the whole body, and becoming confluent; it very closely resembled scarlet fever; the face was involved, here the rash being in the form of a uniform blush; rash lasted twelve days; followed by well-marked desquamation in flakes (scales), which continued for a week; no sore throat or strawberry tongue, and no change in the general symptoms; temperature ranged between 102 and 104.4° F., both before and during the eruption; never had had measles or scarlet fever; was sent to isolation ward.

Case VI.—Female; aged 21 years; prolonged, moderately severe course; on thirty-ninth day, when temperature was 103 to 104° F., a scarlatiniform, punctated, bright-red eruption appeared on the chest, flank, and abdomen; no sore throat; course of disease not affected by rash; lasted two days; no desquamation; never had scarlet fever or measles.

Case VII.—Female; aged 10 years; attack mild; finely punctated erythema on chest, abdomen, and back on eighth day; rash bright-red, disappearing on pressure; face free; lasted five days, and followed by slight, branny desquamation; throat clear; no alteration in temperature at appearance of rash; history of scarlet fever and measles.

Case VIII.—Female; aged 25 years; on admission, on fourteenth day, consolidation of left lower lobe noted; had typical, moderately severe attack of typhoid fever, temperature ranging between 102 and 104.8° F., and normal on thirty-second day; on sixteenth day of disease, a bright-red, punctated rash, indistinguishable from that of scarlet fever, appeared on shoulders and chest, spreading in two days to abdomen and thighs; face of a brilliant scarlet flush; on fifth day, rash fading; next day (sixth of eruption and twenty-second of disease), rash suddenly became more intense and of an entirely different character, consisting of irregular, elevated, dark-red papules, resembling measles most closely; involved the whole body, except face, in successive stages; lasted nine days; desquamated on eleventh day from appearance of rash in well-marked, branny scales; desquamation lasted several days; no sore throat or alteration in temperature or general symptoms at or during rash; history of measles; no scarlet fever.

The patients in this group all exhibited eruptions closely resembling scarlet fever,—that is, a scarlet, or dark-red, punctiform eruption, lasting from two to twelve days, and followed in three cases by desquamation of the affected areas, but never of the finger-tips, as in true scarlet fever. None had sore throat or strawberry tongue, and in none was the general course of the disease affected by the eruption. The rash had its seat in all on the back, chest, and abdomen; and in two, on the legs and arms, also. In three the type of fever was severe, and in one mild. Three were females and one was male. All were under twenty-six years of age. Two

had had scarlet fever in childhood, and two had not. In one case the efflorescence appeared on the eight day, and on the sixteenth, twenty-second, and thirty-ninth days in the remaining three cases, —again exhibiting the tendency of the rash to reveal itself in the middle or late stages of the fever. One of these patients was isolated.

Morbilliform Rashes.—Still another, and a rarer, variety of the erythematous rash is the morbilliform. None of the Johns Hopkins cases were of this type; but J. C. DaCosta has reported two cases, and H. Neumann one. It is to be noted that by morbilliform eruption is not meant an extensive and confluent variety of the specific rose-rash. The latter is not infrequent, while the former is one of the rarest of the adventitious eruptions of typhoid fever. There were two examples of this rash in my serise, as follows:

Case IX.—Female; aged 23 years; attack moderately severe; temperature ranging between 101 and 103.8° F.; admitted on the seventh day; on ninth day, a macular eruption appeared on abdomen, back, and chest; gradually covered whole body, and lasted eight days; composed of elevated, red papules, closely resembling measles, but not crescentic; distinct from specific rose-spots, which were present on admission. No implication of mucous membranes; no alteration in general symptoms; no desquamation; history of scarlet fever and measles.

Case X.—Female; aged 24 years; very ill; delirium and high temperature (105°); normal on twentieth day; on twenty-second day, profuse, papular, non-crescentic, measles-like eruption, livid in color, and in places petechial; limited to abdomen, chest, and back; no alteration of temperature at time of rash; no catarrhal or throat symptoms; rash lasted three days; no desquamation; no history of scarlet fever or measles.

In both cases the eruption bore a close resemblance to measles, both in its color and in its general characteristics, except that it was non-crescentic. It was unattended by catarrhal or general symptoms, other than those of the original disease. In one, it appeared on the ninth, and in the other on the twenty-second, day, lasting three and eight days, respectively. In neither case was it followed by desquamation. One patient had had scarlet fever and measles, and one had not. In both patients the disease was of

severe type. According to reported cases, this appears to be the rule in rashes of this character.

Erythematous rashes are likely to make their appearance at two periods in the course of typhoid fever, viz., early in the disease, at the onset of the fever, and in the middle and later stages of the attack, from the second to the fifth week, and even later. The rashes of the earlier period are usually of the nature of a passing erythema of short duration; they are of the character met with in the initial stages of other forms of pyrexia, and are probably of vasomotor origin. The late eruptions, to which all my cases appear to belong, occur from the beginning of the second week onward, and are of a different nature—the rash being more definite, more prolonged, and of more specific character. Many, if not most of them, are due probably to the absorption of toxins, either from the intestinal ulcers or the fecal contents of the intestines,—that is, they are either of septic or of digestive origin. They are analogous to the eruptions of so-called surgical scarlatina, to the scarlatinal rashes of the puerperium, and to those occurring after the ingestion of certain foods, such as spoiled meats, etc. H. Neumann mentions a case of measles-like eruption supervening on the sixteenth day of typhoid fever, coincident with the appearance of a pure culture of Streptococcus pyogenes in the urine. Nervous influence, without doubt, plays an important rôle in the production of some of the rashes. This was shown, I think, in my two cases exhibiting autographic skins. To drug-ingestion cannot usually be attributed the majority of the rashes attending typhoid fever, as they appear under all kinds of treatment. Osler remarks that he has encountered them more frequently since he ceased to administer quinin, than when he gave this drug in large doses to every patient with enteric fever. The relation of enemas to the erythematous form of rash is very uncertain. Of the 10 cases, 5 had been given simple enemas of soap and water before the appearance of the eruption, and 5 had not. In only 2 of the former did there appear to be any direct relation between the two, *i.e.* the rash appeared within 24 hours after the administration of an enema, and this was the first enema given to the patient; the enema, therefore might well have caused the rash. In the remaining 3, enemas were given during the whole course of the disease, both before, during, and after the

outbreak of the eruption. Each patient, too, had been taking enemas for some days or weeks prior to the appearance of the eruption, so that it is impossible to estimate how much influence the enemas had upon the production of the efflorescence; in my opinion probably none whatever.

Herpes.—There were four patients with herpes among the 250 cases,—that is, 1.6 per cent. In the Johns Hopkins series of 829 cases herpes was noted 29 times (3.5 per cent.), and Zinn met with it in 5 per cent. of 190 cases. It was usually present on admission although the admission was not always early in the disease. The actual date of its appearance was the fifth, twenty-fourth, fifteenth, and thirteenth day, respectively. The eruption was on the lips in the majority of the patients, and was not attended by alteration in the general symptoms. None of the cases was complicated by malaria or pneumonia. In all the fever was of severe type. The cases are as follows:

Case XI.—Female; aged 18 years; moderately severe, prolonged attack; fever lasted forty days; when admitted, on seventh day, had profuse crop of herpetic vesicles on left upper lip and alæ nasi, which had appeared two days before admission; lasted twelve days; no malarial organisms; no pneumonia.

Case XII.—Female; aged 26 years; severe attack; temperature lasting forty-seven days; when admitted, on twenty-ninth day, had several herpetic vesicles, in stage of crusting, at the right corner of the mouth and on the upper lip; had been present three or four days; lasted seven days; no malarial organisms; no pneumonia.

Case XIII.—Female; aged 16 years; moderately severe attack; temperature often being above 105° F., and continuing twenty-six days; admission on fourteenth day; one day later, small patches of herpes appeared over upper and lower lips; lasted about a week; no malarial organisms; no pneumonia, though patient had marked bronchitis.

Case XIV.—Female; aged 27 years; very severe attack; violent delirium and three hemorrhages; temperature from 103 to 104° F.; herpes on alæ nasi (five or six vesicles in stage of crusting) on admission, on fourteenth day; lasted five days; no pneumonia or malarial organisms.

The occurrence of herpes is rare in typhoid fever, especially so

in comparison with pneumonia or malaria. I can recall but few instances. So infrequent is it in enteric fever that Strümpell says that in case of doubtful diagnosis it is a factor in excluding the disease. In one of the cases just related, it was of so prominent a character that pneumonia or malaria was at first suspected. Like the erythematous rashes, it is probably more frequent in some epidemics than in others.

Urticaria.—Of this form of cutaneous disturbance, there were two cases, or 0.8 per cent. Its great infrequency is shown in the Johns Hopkins series, in which it occurred but three times, or in 0.36 per cent.; while among the 350 male typhoids in the Episcopal Hospital during the past four months, there was but one case of urticaria. It was general in distribution in both cases, while this was so in only one of the Johns Hopkins patients. Both of my patients were females, as follows:

Case XV.—Female; aged 43 years; moderate attack; fever lasting twenty-four days; on nineteenth day, urticarial wheals appeared on trunk, and soon covered whole body; lasted three days, and then reappeared more severely after an interval of five days, again lasting three days; temperature during the outbreak, 100 to 101° F., and not affected by eruption; no dietary errors; no enemas given.

Case XVI.—Female; aged 4 years; very severe attack; temperature high (105°); constant delirium; severe nephritis; typhoid bacillus in urine in pure culture; on thirteenth day, profuse urticarial eruption in large and small wheals, some as large as silver dollars; was accompanied by papular eruption the size of large pin-heads; wheals lasted twenty-four hours; papular eruption (urticaria papulosa), five days; no desquamation; patient died on thirty-sixth day; no dietary errors; no enemas given.

F. Woodbury has observed a case in which an urticarial eruption preceded the specific rose-rash in enteric fever, appearing on the seventh day, and receding as the rose-spots came out on the eighth.

The usual origin of urticaria in disturbances of the gastro-intestinal tract probably obtains in typhoid fever, as in other conditions; indeed, when we consider the conditions of this tract in enteric fever, it is, perhaps, remarkable that urticaria is not more

frequent. In the two cases, the influence of drugs, enemas, and shock (nervous influence) could be positively excluded.

The accidental rashes of typhoid fever are of interest largely from a scientific and diagnostic standpoint. This is especially true of those eruptions belonging to the erythematous group. If Case V, cited as typical of the scarlatinal class, had presented sore throat, or had occurred apart from typhoid fever, the diagnosis would have been most difficult. Jenner gives the interesting history of a patient in whom a similar condition was mistaken for scarlet fever. It should be remembered, also, that it is quite possible for scarlet fever or measles to appear in an individual ill with typhoid fever, the two infections pursuing their specific courses side by side in the same patient. I, myself, have met with an instance of this form of double infection. In establishing the diagnosis of scarlet fever, it should constantly be borne in mind that too much stress must not be placed on the character of the rash, or, as Schamberg remarks, on the mere occurrence of desquamation. The eruption of scarlet fever is most variable in its features, and may not differ from other forms of erythema; while desquamation is likely to follow any acute inflammatory condition of the skin, except that in the latter case it is prone to succeed the eruption more closely than in true scarlet fever. To arrive at a correct conclusion, the whole clinical picture must be carefully considered and studied, and even then, the exact diagnosis may remain in doubt. That all doubtful cases should be isolated goes without saying.

The occurrence of herpes ought not to obscure the diagnosis of typhoid fever. Careful examination of the blood and chest will usually exclude malaria and pneumonia, although here again it must not be forgotten that either of these infections may co-exist with typhoid fever.

The adventitious eruptions of enteric fever appear to have no deleterious effect upon the disease; only one of the 16 cases was fatal, a girl of four years, with very grave type of infection.

Note.—Since preparing this paper, there has occurred in Dr. E. J. Morris's typhoid fever wards in the Episcopal Hospital an instance of erythema multiforme, in the person of a young man of sixteen years. The rash appeared on the eighth day of a mild attack of typhoid fever, and consisted of large gyrate forms and

concentric rings of various tints of color. The development was most extensive, involving most of the back, abdomen, and chest. It lasted five days, and then rapidly faded.

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THE IRREGULAR HEART: ITS CAUSES AND TREATMENT

A LECTURE DELIVERED AT THE MEDICAL GRADUATES' COLLEGE AND POLYCLINIC, LONDON

BY BERTRAM ABRAHAMS, M.B., B.Sc., F.R.C.P.

Assistant Physician to the Westminster Hospital; Joint-Lecturer on Medicine in the Westminster Hospital Medical School; Examiner to the Conjoint Board, London, England

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GENTLEMEN: To-day I wish to call your attention to a symptom. In discussing symptoms, one has to consider the fact that any of them may mean very little or almost nothing, or on the other hand may betoken a condition of the utmost gravity.

I think I ought to say at the beginning that, personally, in the examination of the symptom to which I ask your attention, irregularity of the cardiac action, I do not lay very much stress upon recondite methods. What I mean is this, that if one takes a sphygmographic tracing, and applies to it accurate mensuration, it will be found that the tracing is irregular, and shows oscillations, corresponding to the respiratory rhythm, to Traube-Hering curves, and so forth. These have no more bearing upon clinical medicine than the infinitesimal amounts of sugar and of albumin which chemical research can demonstrate in normal urine. The irregularity which I wish to consider is the irregularity which you can hear or feel. No doubt the use of instruments of greater precision may be valuable in its further investigation, but I do not think that much is gained in this way from the practical point of view, and particularly from that of those who examine patients in any numbers.

Before beginning to discuss the question of irregularity of the heart, it is necessary to say a few words as to the cause of cardiac rhythm in general. Rhythm is a very common phenomenon of protoplasm. The respiratory center has a well-known rhythm. As regards the nerve-cells of the spinal cord, if you apply a stimulus to the motor area of the cerebral cortex at the rate of 100 per second

you will not get tetanus in the voluntary muscles related to it; you will only get clonus at the rate of six or seven beats a second, the same rate as that of ankle clonus. This indicates the specific rhythm of the nerve-cells in the anterior cornua of the spinal cord.

What does rhythm mean? I suppose one may say that a certain amount of energy is used up by the protoplasm, and a short interval required for the energy to be replenished, and for recuperation to take place. This necessitates a brief period of rest repeated at frequent intervals, resulting in rhythm. With regard to the heart-beat, the rhythm of the cardiac muscle is not dependent primarily upon any nervous mechanism whatsoever. The fetal heart begins to beat before the nerves are developed. If you take the heart of the tortoise, and by a zigzag incision cut the ventricle into a long strip, so that no nervous connection can exist between its two ends, under appropriate conditions the muscle will continue to beat, although no nerve impulse is possible. The rhythm of the heart depends primarily upon the properties of its muscle, and we must associate disturbance of rhythm above all things with a disturbance of cardiac musculature. That irregularity of the heart in many cases betokens serious defects in the cardiac musculature is a fact of essential importance which may be directly inferred.

In addition to the muscle, a complicated nervous mechanism has something to do with the heart-beat. The relation of nervous to myogenic mechanism is in the nature of a control, or a kind of escapement. We find that it is an almost universal physiologic law that where a mechanism is of the greatest vital importance a double supply has been provided. Take for example the respiratory mechanism. The ordinary movements of respiration are dependent upon impulses coming *via* the vagus from the lungs to the respiratory center, and returning by the phrenics and intercostal nerves. Should this mechanism be thrown out of gear, its work is preformed, not quite as perfectly, to be sure, by the chemical influence by which the center in the medulla is stimulated when the CO_2 tension in the blood is in excess. Similarly, in the case of the heart a second apparatus is afforded by the nerve-supply through the vagus and sympathetic nerves. These are of importance in regulating and fixing the mechanism of the heart-beat, the vagus being both an afferent and efferent nerve, while the sympathetic is efferent only. Reflex action

affecting the regularity of the heart is undoubtedly bound up with the cardiac centers in the medulla, and not dependent upon the cardiac ganglia. These latter are distributing organizations, through which flows all the nervous energy that may be required, but cardiac reflex action depends solely upon the centers in the medulla. This is all that it is necessary to say here with regard to the physiology of the symptom under consideration.

Applying this practically, we find that cardiac rhythm is myogenic in origin, that it is conducted along the muscular fibers, and that it is dominated by the rhythm in the auricles; the nervous apparatus affords a secondary means of control. In considering, therefore, the symptoms of irregularity of the heart, it must be borne in mind that the rhythm of the heart is, as far as we are concerned, essentially independent of the nervous system. Hence a main character to be noticed in respect to the cardiac rhythm is its extreme stability. In a heart beating at the rate of 200 or 240 a minute, or one beating at the rate of 30 a minute, the rhythm as a rule maintains its regularity, being ordinarily unaffected by the greatest variations in the speed of the organ.

With regard to the varieties of arrhythmia, taken from the most general point of view, we shall consider first those which are quite temporary or evocable,—that is to say, those which are brought about by the action of some sudden impulse upon a naturally rather unstable heart. You have all come across instances in which the heart begins to become irregular the instant a stethoscope is applied. Then there are cases in which a fluttering sensation is felt, associated with actual irregularity, if the patient rises suddenly from the sitting posture. There is also that kind of irregularity of the heart which is usually termed paroxysmal tachycardia, in which a few rapid beats may be interspersed amongst the normal ones. This may be produced by fright or a variety of similar causes. In gastric neurasthenics it is not uncommon to find irregularity of the heart associated with a slow pulse (bradycardia). You all have probably had the opportunity of observing a similar phenomenon in normal children. When they are taking a deep breath, the heart frequently becomes irregular, and the pulse slows. This has been observed in certain pathologic conditions, and has been dignified with various Latin names, but it is only an exaggeration of what

is normal and physiologic. Then there is an irregularity of the heart which is a little more persistent, which may come on under the influence of certain external or internal circumstances, and then pass off as if it had never occurred. The most conspicuous instance of this is the irregularity which frequently occurs at the menopause. In these cases you may find a cardiac irregularity lasting for eight or ten months. Again in some intoxications, and in certain reflex irritations, such as dyspepsia, there may be a prolonged, but not permanent, irregularity of the heart. Lastly, there is permanent irregularity of the heart, which may or may not be serious, but which should always make one suspect something wrong with the organ itself.

The discussion of the degree of irregularity is I think a matter upon which one may waste a good deal of time. Of course all sorts of pulses have been described, and picturesque diagrams made of them. I myself have taken a large series of tracings, but I really think that one does not learn very much of practical utility from them.

Here one may mention that it is well recognised that there are people who have an intermittent pulse all their lives. A most notable example of this was the great Talleyrand, whose pulse dropped a beat after every three, and who said that he expected to live a third longer than most people on the strength of it. As a matter of fact I believe he did so. One may call this kind of thing a typical abnormality, which goes on constantly, and which will never be different in any way. It may be added that, except when it occurs in the course of cerebral disease, intermittence is in general a less dangerous phenomenon than irregularity.

Another variety, which is often of little importance, is that in which the patient has a heart which now and then, at no regular intervals, drops a beat. Neurasthenic people are likely to have this variety of irregular heart and intermittent pulse. A patient of mine was disturbed almost every night when going to sleep by the pulse dropping one or two beats. He woke up, and then fell asleep normally without any further trouble. These cases usually are not very serious.

Then you may meet with a regular irregularity of the heart, as one may term it. The patient's heart may beat at one rate for some

little time, and then the rate may suddenly change and so on, in a cycle. This has been dignified by German authors with the name of "allorhythmia."

Other forms lead up by stages to that of excessive irregularity of the heart. Here there is a kind of pulse in which one beat tumbles over another, and in which no two beats are of the same length, or of the same strength, or have the same interval between them. This is almost invariably a sign of myocardial degeneration.

We may here go over the etiologic characteristics of irregularity of the heart, as upon them I wish to base the views which I hold as regards treatment. Irregularity of the heart may be produced by three different groups of causes; that is to say, by affections of the heart-muscle, affections of the nervous mechanism, and reflexly from affections of the distant organs. These three groups include every cause of irregularity of the heart which is of real, practical, importance. It may be produced by various other causes, but they are of no moment.

Irregularity of the heart owing its origin to affections of the cardiac muscle is the most important variety, not because this is the most frequent cause, but because it is the most dangerous, and requires the most careful treatment. Primary irregularity of the heart always indicates cardiac insufficiency, resulting in the heart being unable to do its work properly. There may be two reasons for this: (1) failure of the heart-muscle; (2) the fact that the heart has to do work which is too much for it. The insufficiency thus may be actual, or relative,— actual, as in myocarditis, or relative, as in arteriosclerosis. As regards the relative form, it has been definitely shown by experimental evidence that if the peripheral blood-pressure and peripheral resistance are raised beyond a certain point in any animal, the heart invariably becomes irregular.

Let us now investigate more closely the causes of true insufficiency of the heart. They are as follows: (1) Intoxication, or poisoning of the heart muscle; (2) inflammation of the heart, such as pericarditis, endocarditis, and myocarditis; (3) fatty degeneration of the heart; (4) fibroid degeneration of the heart; and (5) old valvular disease,—for toward the end of obstructive heart-disease, unless death is sudden, the heart is almost always irregular. These are the main causes, and they are all causes which tend to act

upon the heart-muscle. As a matter of fact, we live upon our heart-muscle, and that is why an irregular heart may be the first warning of the approach of death.

As regards intoxications, you know perfectly well that the excessive use of tobacco, juvenile smoking especially, excessive coffee-drinking, or the drinking of green tea, may cause a persistently irregular heart. Digitalis, aconite, belladonna, and various other drugs, may produce the same symptom. Certain infective diseases can also, by means of their toxins, lead to irregularity of the heart. The chief of these is diphtheria, and amongst others one may mention pneumonia and acute nephritis. The appearance of irregularity of the heart in the course of an acute infection is a sign of the greatest importance, and one which must be treated immediately.

Of inflammatory affections of the heart pericarditis is one of the commonest, and there are very few cases of pericarditis in which irregularity of the heart is not present. Years ago it was demonstrated by Heitler that if all the nervous connections of the heart be destroyed, and the pericardium irritated or stimulated in any way,—by hot water or scratching, for instance,— irregularity of the heart will ensue. The incidence of pericarditis in the course of attacks of rheumatism is often indicated by irregularity of the heart. Of course the irregularity is to be detected by the ear, because it is not always possible to diagnose irregularity of the heart by the pulse, as this may be apparently regular whilst the heart is irregular. This is particularly the case when the force of successive beats is unequal, the weaker ones not being transmitted to the periphery. Endocarditis and myocarditis also cause irregularity of the heart, though perhaps less often than does pericarditis.

With regard to the fatty heart my experience is that irregularity from this cause is not usually seen in young subjects, or if it is, the irregularity is slight and easily curable. In people over fifty, however, this irregularity is apt to be persistent, extreme, and very dangerous, frequently leading to a fatal termination. It may even result in that interesting condition described by MacWilliam as "delirium cordis," in which the normal rhythm of the heart has disappeared altogether, and there is only muscular peristalsis, over which, as regards the ventricle at any rate, the vagus has lost all power of control.

Fibroid degeneration produces irregularity of the heart, which in many cases is very pronounced. The symptom may be one of the first indications that the heart has become deranged in this fashion.

In valvular disease, toward its termination, there may be a time when the murmurs disappear and the heart becomes irregular. This is the worst possible period in a heart case. Many of these cases are not seen in consultation until something of this sort has developed, and when it has reached such a stage that the fluid-vein in the heart is insufficient to produce a murmur, while at the same time the cardiac muscle is so weak as to make the beat irregular.

With regard to causes arising elsewhere than in the heart itself, there are of course many affections of the central nervous system in which the heart is apt to become irregular, such as meningitis, cerebral hemorrhage, and also certain psychical states. Again, in cases of pressure on the vagus irregularity of the heart is often present. I have seen it caused in this way by enlargement of glands. It is said that the symptom may arise from disease of the cardiac ganglia, though no case of this kind has come under my own observation. Reflexly, of course, almost anything can produce irregularity of the heart. Laryngeal stenosis frequently causes it; it also occurs in diseases of the bronchi, diseases of the liver, and diseases of the stomach, the latter being the most common of all such etiologic factors. The symptom may also appear in disorders of the kidneys, or almost any of the organs of the body, but is much the most frequently seen in association with affections of the stomach; in this connection it will readily be remembered that, no doubt owing to vagus association, the paroxysm of true angina pectoris often ends with flatulent eructations.

In diagnosis the first object is to find out whether we are dealing with a case of primary cardiac disease or not; if not, nervous and reflex causes must be successively considered. As regards this the quality of the first sound is the best and most reliable guide, and gives one most information as to the muscular power of the ventricle.

It is interesting to observe that arteriosclerosis may lead to irregularity of the heart in one of two ways,—either by causing increase of tension, which I said just now has been experimentally

shown to produce an irregular heart,—or by affecting the coronary arteries, and causing cardiac degeneration. In the first instance, if the irregularity is simply produced by high tension, the heart being virtually normal, you will have a good, or even accentuated, first sound. Should the irregularity be produced by the second cause mentioned, the first sound will tend to be weakened or blurred. The first thing to be done is to ascertain the quality of the heart-beat and the loudness of the sound, after which one should examine for arterial symptoms. Associated neuroses should be considered, though not in the first instance. Personally, in these cases I should rather diagnose anything than an irritable nervous heart, and I believe that if the number of "nervous hearts" we have to deal with were investigated with sufficient care, it would be considerably diminished. The diagnosis of functional cardiac disease should only be made by rigid exclusion.

Of course, one ought to examine the heart in the first place as regards the condition of the valves and the freedom of the circulation through the organ. Then other organs should be examined, especially the stomach, in order to ascertain the condition of the digestive functions. The nervous system should be investigated from the point of view of some gross lesion in it causing the irregularity of the heart, and not till all these have been negatived should the possibility be considered of the irregularity having a reflex origin, or being produced by a psychosis.

In the diagnosis of these cases one is, of course, largely influenced by the quality of the pulse, which tells us at the same time something with regard to the state of the heart and something as to peripheral resistance. This is a most useful guide in treatment and prognosis. One thing I must mention, which is sometimes rather perplexing. That is that a patient may have organic disease of the heart associated with functional irregularity. It is only to be expected that a heart organically diseased should be more excitable than normal, and now and then people with chronic heart disease have neurotic arrhythmia.

I saw such a case recently. It was that of a lady who had had a sudden cardiac attack whilst going upstairs, and was found by her maid lying on the stairs, with a fluttering, palpitating heart. She was well known to have suffered from heart disease for some years.

I saw her some hours after the attack, when it was perfectly obvious that she had quite recovered from it. The doctor who had seen her first had given bromides, and the result was the disappearance of all functional symptoms. There was at the same time functional weakness of one limb, which helped to make the diagnosis easy. It must not, however, always be inferred that the irregularity of the heart is functional, because the case may be one of heart disease, with the arrhythmia due to myocardial degeneration. Here the diagnosis will mainly depend upon the presence or absence of signs of failing compensation.

As regards the *prognosis,* those cases in which the patient is conscious of the irregularity are usually those in which the outlook is the most favorable. Thus, if you discover irregularity of the heart without the patient's having previously noticed it, the prognosis is worse than if the patient finds it out himself. This is a good rough-and-ready general rule. Moreover, the prognosis is worse when the irregularity of the heart supervenes as a complication of another disease, such as an infection or an intoxication, or if it appears in advanced valvular disease. Of course in purely functional cases the prognosis is not bad at all, and the symptom may persist for a long period of time. I have under my observation at the present time a Dutch lady, who has had irregularity of the heart for a long time, the symptom having been very pronounced during the last four years. She has never had a murmur, and the heart sounds are perfectly clear. The heart does its work well, but there is persistent irregularity and she is frequently sensible of palpitation. In this case I think the nervous system is responsible for the occurrence of the symptoms, and that there is no organic cause for them.

Finally with regard to *treatment.* There are some cases in which the irregularity may persist for years, and only be detected accidentally; it may resist all treatment whatsoever, and yet be perfectly insignificant; that is to say, it may make no difference to the patient's health. The case which I have just quoted is one of these, and several others have come under my observation, in which persistent cardiac irregularity has meant nothing. I am not speaking of cases merely of regular intermittence, but of real irregularity of the heart. On the other hand, one comes across cases—perhaps

more frequently—in which irregularity of the heart is a "cloud no bigger than a man's hand," and appears as a presage of the breaking up of the cardio-vascular system.

The treatment of the functional cases is fairly simple. Bromides are the best drugs, and of the bromides I think that strontium bromide is the best. The action of hyoscyamus is now and then beneficial. I am not very confident about this drug myself, but think that on the whole its effect is good. I do not think that I have ever given it for irregular heart except in conjunction with bromide. Occasional doses of chloralamid or veronal act efficiently as hypnotics. These drugs are useful, because the fluttering heart, which, like all nerve symptoms, becomes worse in the evening, frequently causes sleepless nights, and if, by the employment of veronal, in which I am a great believer, or of chloralamid, one can break the habit of bad sleeping by one or two nights' good rest, one very often, so to speak, turns the corner of this distressing, but not dangerous, condition.

If the heart-muscle is affected the best remedy in all cases, except when there is arteriosclerosis, is digitalis, and this drug may very often be given as a test as to whether the patient is likely to improve or not. If there is a reaction to digitalis, the prognosis is very much improved. But it may be and very often is the case that one is dealing with hearts in which the muscular power is insufficient to cope with the resistance in the peripheral circulation, and in these cases the accentuation of vasomotor power by the employment of digitalis may be dangerous. I have several times seen such a heart, in which steam was got up for two or three days under the influence of digitalis, end by an anginal attack. I remember the case of a lady who had had heart disease for some years. Her doctor came to tell me that she had suddenly ceased to pass urine. He could hear no murmur, though she had previously had a double mitral bruit, and she was evidently very ill. I had a severe cold at the time and it was late in the evening, and I said he had better give her digitalis. In the morning he rang me up to say that she was very much better, and that she had passed five pints of urine. I went to see her, and said that it was just what I had expected. In the afternoon I was again rung up, with the news that she had angina pectoris, which she had never had before, and which was undoubt-

edly due to my treatment. Under strophanthus and iodide she speedily recovered.

That case has been a lesson to me ever since that we should carefully gauge the recuperative power of the heart by every means in our power before giving a drug like digitalis when the circulation is obstructed.

I consider that a safer line of treatment in such cases is to give, if necessary, a diffusible stimulant, such as ammonia and ether, and to follow it with nitrites and diuretin. I am a great believer in the employment of diuretin in these cases. It is a salicyl compound of caffein, and caffein is not only diuretic but has a distinctly stimulating effect upon the heart. Some little stimulant should be given, and in the course of the day 5-grain (0.3 gram) doses of diuretin, repeated four to five times.

If you do this you will probably succeed in draining off the obstructive fluid, and you can then call upon the kidneys to do more of their work. This is the best indication that I know of for this special drug.

I need not go further into the use of iodides and nitrites. It has been shown quite recently that the particular value of iodides in these cases consists in increasing the fluidity of the blood. There is no doubt that the actual thing that iodide does is to increase the plasticity of the blood, and thus lessen the amount of work that the heart has to do. It does not, as was once thought, dilate the peripheral vessels, but increases the fluidity of the blood in much the same way as it increases the fluidity of sputum. This is the rationale of the use of iodide, to lessen friction and resistance in the vascular system.

As regards cases in which irregularity of the heart supervenes in the course of an infection, what are we to do if a patient suffering from enteric fever, pneumonia, or diphtheria develops irregularity of the heart?

In diphtheria irregularity of the heart may proceed from two causes. One of these is degeneration of the vagus; the other, and even more dangerous, degeneration of the myocardium. Now we know that the diphtheria toxin has a very marked action on the heart muscle, so that an irregular heart in diphtheria is a matter of grave significance, and in these cases one has to resort to vigorous

measures. I am inclined to give strychnin, and also inhalations of oxygen. I consider the latter drug to be a most valuable cardiac tonic, though I am not much of a believer in it as a means of providing oxygen for the lungs. In some instances it may be necessary to give hypodermics of ether. There is in fact no remedy which I should not attempt, with a view to tiding the patient over the period of danger. In diphtheria this is very often merely a question of a few hours, and if you can succeed in galvanising the patient into life for that space of time, he will recover.

Lastly, cases often come under observation in which, after many years of cardiac obstructive disease, the physical signs disappear, leaving irregularity of the heart. Here one is in the presence of one of the greatest dangers which ever threaten a sufferer, and in the majority of these cases the prognosis is extremely bad. I believe that counter-irritation is of great value, either by means of leeches applied over the precordium or the liver, or blisters over the precordium. At the same time I am inclined to let off as much fluid as possible by the use of diuretin, and to limit the amount of fluid which the patient takes, though I do not do this in cardiac cases generally. As a matter of fact, there is no doubt that in many cases fluid is valuable in replacing a certain amount of waste through the skin and kidneys, and is as valuable as a mild saline injection. In many cases of irregularity of the heart it is advisable to give a certain amount of fluid, but in those just described I am inclined to restrict it, because I do not believe that the heart is strong enough to drive any increased amount of fluid through the peripheral vessels.

This is all that time will permit me to say with regard to these irregular hearts, but I hope that you will see my point of view with regard to the investigation and treatment of a symptom which may mean a great deal or which may mean nothing, but which you may find will help you to foretell disaster, and possibly avert it.

THE DIAGNOSIS OF SOME CHRONIC JOINT AFFECTIONS

A PAPER READ BEFORE THE WIMBLEDON AND DISTRICT MEDICAL SOCIETY

BY H. S. CLOGG, M.S.(LOND.), F.R.C.S.(ENG.)

Assistant Surgeon to the Charing Cross Hospital, and Surgeon to the Evelina Hospital for Children, London

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The title of this paper, which I have called "Some Chronic Joint Affections," perhaps needs some preliminary explanation. In speaking of a chronic affection, I am not measuring by months or years or any other time limit, but refer to varieties of arthritis which may commence acutely, subacutely, or in an insidious manner. But whatever the onset may be, they tend to become chronic unless treated. Perhaps a better term would be "non-suppurative arthritis." I have prefixed the word "some" because, in the present state of our knowledge, it is impossible to give any accurate classification of cases of non-suppurative arthritis. We have such names as chronic rheumatism, rheumatoid arthritis, rheumatic gout, arthritis deformans, and osteoarthritis, all applied very loosely. Some of these are applied to the same diseases; perhaps some represent different diseases. Just a glance at these names will at once show how treacherous they are.

What justification is there for the name chronic rheumatism? Acute rheumatism is undoubtedly a disease produced by a definite organism, but this organism is still under dispute, and until this point is settled we have obviously no right to talk of a chronic rheumatism. Rheumatoid arthritis, meaning an arthritis of the nature of a rheumatism and yet something differing from it, is obviously meaningless. Rheumatic gout, implying a combination of rheumatism and gout, is certainly not permissible in the present state of our knowledge. Better than these are the names arthritis deformans and osteoarthritis. The former may be applied to any deforming disease of a joint in which the etiology is obscure. The latter, osteoarthritis, should be reserved for those cases of arthritis associated with bony and cartilaginous changes in the joint. These two terms

from an etiologic point of view have nothing to commend them, but nevertheless they are less misleading than such terms as chronic rheumatism, rheumatoid arthritis, and rheumatic gout. I am not going to attempt any new classification of joint disease, but make these remarks more as an apology for avoiding these popular names as far as possible.

A frequent form of joint disease, which is variously called by one of the above names, and for which I prefer the name of osteoarthritis, is characterised pathologically by wearing away of the articular cartilage, some atrophy of the bone ends, some osteophytic outgrowths around the margins of the joint, and, in addition, some changes in the ligaments and synovial membrane. The most characteristic changes in this condition are the bony and cartilaginous changes. Clinically this is evidenced by pain, stiffness, deformity, some grating on movement of the joint, and some irregular thickening of the ends of the bones, which is generally referred to as "lipping of the bones." This condition is well known. But all chronic joint affections are not of this type. In a number of cases, as shown clinically and by a radiogram, there is no change in the articular cartilage or in the bone ends. It is tempting, therefore, to make a classification into those which exhibit bony and cartilaginous changes and those which do not. I do not think, however, that this is accurate. I have seen a picture in which the two conditions are present in the same patient. Whether the bony or cartilaginous changes are the later results I am not prepared to say, but one thing appears evident, and that is that in some cases of chronic joint affection there are no changes in the cartilage or bone after some months or even years, a condition of things which is not so in a typical osteoarthritis. The latter name cannot be strictly applied unless there be erosion of cartilage and osteophytic outgrowth, as shown clinically by the bony grating on movement of the articulation and the "lipping" of the bone ends, or by a radiogram.

I will not discuss the etiology of osteoarthritis. The cause of these other joint affections is often, perhaps in the majority of cases, it may be in all, some infection. They are examples of infective arthritis. By infective arthritis is understood an arthritis set up by some microörganism or the toxins of a microörganism. The diagnosis of an infective arthritis deals with the recognition of the

primary source of the infection. This can be traced in some cases, as I will point out shortly. I am not denying that osteoarthritis may be infective in origin also, but there is lack of evidence on this point. It may yet be proved to be an infective arthritis.

Bacteriology is a comparatively young science. There are many obstacles in the way of a scientific investigation of these joints, and the isolation of organisms from the joints is very difficult and has only been accomplished in a comparatively few cases. But gradually our knowledge is being extended and fresh facts are brought to light which tend toward showing that many cases of chronic joint disease are really of the nature of some infection.

The difficulties in the bacteriologic examination of joint diseases may be appreciated if we consider for a moment that common disease, rheumatic fever, a disease known for many years. It was so long ago as 1875 that Klebs isolated microörganisms from the cardiac valves in this condition. In 1887 Popoff obtained cultures from the blood of rheumatic subjects, which on inoculation into animals gave rise to pericarditis, endocarditis, and arthritis. Since then many different organisms have been associated with rheumatic fever, and the tendency became to regard it as due to a variety of agencies. Later researches, particularly by Paine, Poynton, Beaton and Walker, have tended to show that in a number of observations upon rheumatic patients an organism can be obtained. This organism isolated by these observers appears to be a micrococcus of some sort; whether the ordinary streptococcus or some specific micrococcus, is not yet definitely settled. Experimenting with the coccus, it appears to be capable sometimes of inducing suppuration, sometimes acute non-suppurative arthritis, and sometimes produces a more chronic inflammation of the joint. It may be then that the same organism may set up various changes in the joints, depending on its virulence. Hence chronic rheumatism may be a justifiable term. On the other hand, many observers have failed to find any organism associated with rheumatic fever. Cole for three years examined many cases of rheumatic fever, the joint effusions and the blood bacteriologically, and was unable to isolate any organism. Philipp investigated 26 cases of acute rheumatism in Pibram's clinique. Cultures were made from these upon a number of different media, and inoculations made into guinea-pigs, dogs, monkeys, and calves. In not a single

case did he get any growth on the culture media. If we have such varied results given by different observers in the common condition of rheumatic fever, it will be readily appreciated how difficult is the bacteriologic examination of chronic joint affections. It is most unusual to have an autopsy upon these patients, and when they die, from some intercurrent affection, the joint disease has been so long in existence, that the organism, if it were one originally producing the disease, will in all probability have died, having left upon the joint its permanent and crippling defects.

Experimentally by inoculations various joint affections may be produced. Some recent and interesting investigations upon this point have been made by Cole. He made cultures and inoculations from six races of streptococci. The sources of these races were: Peritonitis, following carcinoma of the stomach; puerperal fever, autopsy; blood during life in terminal septicemia; empyema; scarlet fever; adenitis. With all these races studied, intravenous inoculation of amounts insufficient to cause the death of animals, led to the production of mild grades of arthritis. On the fourth or fifth day the rabbit appeared ill; it remained quietly in the corner of the cage, and only when urged to move would it do so, and then it was noticed to be quite lame. Usually recovery from the lameness occurred in a few days. In some cases death occurred. Others were allowed to recover and were then reinoculated, and would again show signs of arthritis. One was kept reinoculated in this way over a period of seven months. It appears from this that many varieties of organisms would be capable of producing arthritis, and if the poison were sufficiently attenuated and absorbed for a sufficient length of time, might result in a chronic polyarthritis.

Some labor has been expended in the bacteriologic examination of cases of chronic polyarthritis. As mentioned before, autopsies on such subjects are few and far between, and in addition to this, owing to the small effusion in these cases, it is a difficult matter to aspirate any for examination. Nevertheless in some instances positive results have been obtained. I will briefly mention a few. In 1896, Bannatyne, Wohlmann, and Bloxall, in a series of cases of what they described as rheumatoid arthritis, were successful in some in isolating an organism. This is fully described in their paper. Their conclusions were that the disease was due to the presence of microörgan-

isms in the joint fluids and tissues. How the organisms gained access is not known definitely, probably through some catarrh of the gastro-intestinal or genito-urinary tract. In not a few cases the infection had been traced to the tonsils with almost certainty. Gask describes the case of a woman, aged twenty-nine, who was delivered of her second child. The labor was apparently normal and not followed by fever. A few days later the right knee became swollen, hot and red; subsequently many joints were similarly affected and the patient was confined to bed. The appearance of the patient was one of acute polyarticular, rheumatoid arthritis. Both knee-joints and ankle-joints were slightly swollen, painful and stiff; the wrists, carpal, and metacarpal joints were in a similar condition. Salicylates were given without relief. Bacteriologic examination in films and cultures showed the presence of a streptococcus not to be distinguished from Streptococcus pyogenes. This streptococcus when injected subcutaneously into mice and intravenously into a rabbit proved to be non-virulent.

Hale White records a case of a girl, aged twenty years, who had had several attacks of polyarthritis, lasting some months. All the joints of the hands, both wrists, elbows, and shoulders, both hips, knees and ankles, and as far as could be made out the joints of the feet, were fixed, most of them completely. The temporomaxillary joint and the spine were also affected. The swelling appeared to be as much a general thickening around the joints as an excess of fluid in them. Nowhere was there the slightest evidence of bony outgrowths. This patient died, and from one of the knee-joints a microörganism was obtained.

Poynton at the necropsy of a man who had died from carbolic-acid poisoning found that several of his joints were crippled with chronic destructive arthritis. The structures of the articulation were much diseased, the synovial membrane was much thickened and in places hyperemic; the cartilages were destroyed in places and the bones eburnated at the points of pressure. From the synovial membrane he obtained a growth of diplococci and of Bacillus coli. Experimenting with these organisms, he was able to induce a polyarthritis and from the affected joints isolated the diplococcus. He was also able to obtain an experimental osteoarthritis, with erosion of cartilage and bone. He concludes therefore that the diplococcus

was the cause of the destructive arthritis in the patient from whose joint he obtained it.

There is evidence clinically to show that an arthritis may be set up by the injections of antitoxic sera. Menzer obtained an anti-rheumatic serum for therapeutic value, and in some cases arthritis recommenced as the result of the injections. Arthritis has also been observed after the injection of Koch's tuberculin. Pain in the joints and some swelling have been noticed after the injection of diphtheritic serum, and it has also occurred with antistreptococcic serum. These are very interesting facts, seeming to show that arthritis may be set up by the toxins of bacteria, without of necessity the organisms themselves gaining access to the joint. This adds to the evidence that an arthritis may be of an infective nature, and yet of necessity organisms may not be in the joints. In connection with the acute specific fevers, joint affection has long been known. In scarlet fever it is not very unusual, and is often called rheumatism. There can be little doubt, however, that it is an infective arthritis produced by the organism of scarlet fever, whatever that is. In typhoid fever a synovitis or arthritis is occasionally seen; it may be a mild and evanescent synovitis, or a more chronic form resulting in some ankylosis, or occasionally a severe suppuration may occur. Similarly in pneumonia, erysipelas, and influenza the joints may be involved. In connection with influenza, Witzel gives some very remarkable figures, which I think most would dispute. He says that in about six per cent. of cases there is serious joint disease. Howard Marsh has recorded two cases of influenzal arthritis, in a man aged forty-two years and a boy aged eighteen. Some three years ago I saw a lady, about thirty-five years of age, who some months previously had had an attack of influenza. During her convalescence she developed pain in her left knee, which became swollen. During this six months the pain and swelling in and around the joint persisted and varied in severity, but at all times was so bad as to confine her to bed. She also suffered from irregular febrile attacks, during which the knee would become more inflamed. The swelling was more around than actually in the joint and extended for some distance beyond the real outline of the joint cavity. The synovial membrane was considerably swollen; there was very little increase of fluid in the joint. There was no change in the cartilage or bone. There can be very

little doubt, I think, that this is an example of influenzal arthritis. The main features of this form of arthritis are its chronicity, its usual limitation to one joint, and its tendency to end in fibrous ankylosis and permanent stiffness.

Apart from this arthritis in association with the acute specific fevers, there are other sources of infection from which the joints may become involved. First, I wish to say a few words about gonorrheal arthritis. Gonorrhea attacks joints in various ways. The acute or subacute variety which comes on during the height of the gonorrhea, or during its decline, is usually sufficiently obvious, and the diagnosis is usually perfectly easy. It is the more chronic cases which occur during a gleet, which is never mentioned, that present the difficulties in diagnosis. It may be said that an arthritis may develop at any time during the course of a gonorrhea or gleet, and it may occur in those persons who have a gleet but are unaware of it, and in fact who emphatically deny it, although perhaps admitting a gonorrhea contracted some months previously. In one or two cases recently I have had the opportunity of proving this. This is such a case:

A man, aged forty-two years, came complaining of his left foot, which had been swollen and painful for a few weeks. On examining this, there was considerable puffiness around the tendons on the inner side of the ankle-joint and around the astragaloid-scaphoid joint on the inner aspect of the foot. The arch of the foot was flattened. He complained of some pain in the heel also (a common point of pain in gonorrheal rheumatism). I discovered that his right knee was swollen and hot, but very little painful or tender. This had been so for four months, and came on during the decline of a gonorrhea. He had at this time no symptoms of urethritis, and I was unable to see any discharge from the urethra. There were, however, distinct prostatic threads in the urine, and on rectal examination his prostate was slightly enlarged and his vesiculæ seminales distinctly palpable. This I regarded as secondary to gonorrhea, and the joint affections as due to absorption from this focus.

The fact that I wish to emphasize is that an arthritis may arise at any time during the course of a gleet, and that the latter if not carefully looked for may pass unnoticed; that it is always one's duty in any case of arthritis most thoroughly to examine the urethra and

exclude this as a source of infection. It may be that with a more careful examination this will be found to be a more frequent channel of infection. A previous history of gonorrhea is often obtained in patients afflicted with chronic polyarthritis.

One must never too readily admit that an injury may be the sole cause of a synovitis. That an injury may determine an infection of a joint is undoubted, but often this is only the predisposing cause. In support of this, I can remember two cases, the one a "shoulder" and the other a "knee," said to have followed some injury. Aspiration of the joint showed the gonococcus in the fluid.

Another fairly frequent source of joint infection is the female genital tract, and this apparently apart from a gonorrheal inflammation. The common vaginal and uterine discharges are responsible for some cases of infective arthritis.

A typical instance is as follows: A patient, aged thirty-three years, complained of pain and swelling of the left hand. On examination of this hand, there was some puffiness around the wrist and some of the metacarpo-phalangeal joints. The disorder in the wrist began acutely, that in the other joints more chronically. For four years this patient had had some vaginal discharge, following delivery. The joints had been affected for only a few weeks. Occasionally the temperature was slightly elevated. Under treatment directed to the vaginal discharge the joints considerably improved. A relapse followed a few weeks later, and appropriate treatment again resulted in considerable improvement in the joint inflammations.

A second case is that of a woman, aged thirty-eight years, who for nearly two years had had a swelling around the wrist joints. The onset in these joints was subacute, with pain, tenderness, and some swelling. The hands were rendered more or less useless for a time. Six weeks before the joint inflammation she had been delivered of a child. The labor and puerperium were normal. There was no puerperal fever. But she had had some leukorrheal discharge from this time. The joints, which at first were regarded as rheumatic, continued to pain; they passed into a chronic condition. At times they were much better and the right wrist came very near to normal, but would occasionally swell up with pain and tenderness. This condition had been going on for about two years, and every

form of treatment had been tried. There were no bony or cartilaginous changes shown by a radiogram. Around the left wrist there was a considerable puffy swelling, the ligaments had softened, allowing of some excessive mobility, and rendering the hand more or less useless.

More histories of such cases could be given, but these two suffice to emphasize that the female genital tract is a not very infrequent source of infection and one frequently overlooked in the examination of cases of chronic polyarthritis. It may be said that almost any septic focus may be capable of setting up an infective arthritis, and I should like to indicate a few further sources whence the infection may arise.

The nasal mucosa may be responsible. A patient, about thirty years of age, had suffered from what she called "the rheumatics" for some few weeks. She also had a mucopurulent nasal discharge. On examining the nose, there was a condition of subacute rhinitis and a small septal ulcer. The joints implicated were some of those of the hands, a wrist, and a knee. The pain in these joints was comparatively slight; around some there was puffiness. The joint symptoms in this case were comparatively slight and in a very early stage, and with the improvement in the nasal condition almost entirely subsided. No doubt if the condition had been allowed to go on untreated, the joints would, as in other cases, have passed on to the chronic stage.

Macalister, in a report of six cases of infective arthritis, gives three of these as being of nasal origin, and he points out that it is not necessary to have ulceration of the nose, though he has seen this. It is quite sufficient to have a swollen mucosa, with retained secretion which may be absorbed.

Oral sepsis is regarded as a source of infection. No doubt this may be a common source. I have not seen a case which I could directly attribute to the mouth. An unhealthy throat, enlarged tonsils, and adenoids are given as sources. The gastro-intestinal tract is always a possibility. In chronic dysentery arthritis is known, and in some cases of arthritis intestinal absorption is suggested as being the cause.

The fact that no organism can be detected in the joint does not of necessity show that an organism is not the cause. The difficulties

in the bacteriologic examination of joints have been pointed out, and again it may be the toxins and not the organisms themselves which have caused the arthritis. Some reliance must be placed on the clinical evidence of these cases.

The joints in the cases which I have been describing as infective arthritis show the following features in general: The swelling is more in the structures around the joint, and in the synovial membrane, than an actual effusion into the joint. Sometimes an excess of fluid may be present in the joint. The puffiness extends some little distance from the joint. There is no bony or cartilaginous change as shown by a radiogram.

Clinically the onset may be acute, subacute, or even quite insidious in some cases. If acute or subacute, they very much resemble rheumatic fever and are called rheumatism. The degree of severity varies with the intensity of infection. Salicylates are given without response. The joint symptoms quiet down, become chronic, always subject to exacerbations, tend toward stiffness, permanent swelling, and often deformity. Fever at the onset and during the joint exacerbations is common. Such a clinical history strongly suggests an infective process. Blood changes have been described, as also enlargement of the lymphatic glands and spleen, as in other infective processes. The source of infection in such cases is by no means always obvious. I have tried to point out some conditions which have come under my experience. There may be other sources, and every attempt should be made to trace them, for it is hardly necessary to point out that it is only by treatment in the early stages that complete cure could be effected. One cannot help feeling that, as more care is extended in this direction, more and more cases of chronic polyarthritis will be found to be infective in nature.

A typical tuberculous joint does not often present any difficulties in diagnosis. Its onset, course, and symptomatology are more or less definite. Its frequent occurrence in children, less often in adults, is noteworthy. But one must never forget that it is not so very infrequently encountered in elderly subjects, and in fact it is one of the more frequent manifestations of senile tuberculosis. The wrist- and knee-joints I would single out as being the more common ones affected at this time of life. The characteristics of a tuberculous joint we are always led to believe are as follows: Its general

limitation to one articulation; pain is usually the earliest sign, then follow deformity and some wasting of the muscles around the joint; some swelling appears early, especially if the joint be superficial, and even in the deeper joints, as the hip, there is some general puffiness around very frequently. When such signs are present, in a child particularly, we invariably think of tuberculosis. The temperature is sometimes not elevated; usually a slight rise of half a degree or a degree may be noticed at some period of the day. Should the further changes of degeneration and liquefaction occur in the tuberculous granulation tissue, then caseation, the formation of a chronic abscess which approaches the surface, becomes septic and breaks through the skin, are all changes quite familiar. Such are the usual signs of a tuberculous joint which is allowed to run its course. But departures from the normal are frequently seen. One is accustomed to associate a tuberculous joint with chronicity, but occasionally we meet with a quite acute onset, and this acute onset is of more frequent occurrence in the hip than elsewhere, and is often attributed to an injury.

The history of an accident should always be taken with some reserve. It is very tempting to regard many conditions as the result of some injury, and too often patients are only too willing to refer the commencement of their symptoms to some injury, imaginary or otherwise.

We should always be careful how we accept the history and not overlook some serious condition by being thrown off our guard by an indefinite account of some fall, or twist, or something of that sort.

On the other hand, in a typical, chronic, tuberculous joint, with an insidious onset, pain is often very variable and at times very slight, or even may be said hardly to exist at all. This is quite common and perfectly familiar in the knee. In this condition the physician is generally consulted because the joint is swollen and deformed. The knee-joint in the child is one where the synovial membrane is affected first, and hence the swelling; some softening of the ligaments occurs, and hence the deformity of backward displacement and outward rotation of the tibia. The joint becomes swollen and flexed; the child limps about without much pain. In the adult, where the bone-ends are more frequently involved, pain is generally marked from the beginning. In the elbow a similar state of affairs

is often present, the pain being very slight as the disease is more or less limited to the synovial membrane. In the hip-joint pain is more commonly present from the beginning, but occasionally one meets with cases in which this symptom is very much in abeyance, the patients "limping about," the limp being not so much dependent on pain as the deformity of the joint. In the shoulder the tendency to suppuration is slight. It is the dry form of caries which is generally seen in this articulation, the signs being pain, fixation of the joint, and wasting of the surrounding muscles. This joint is comparatively infrequently involved in the child, and rarely but comparatively more frequently in the adult. For the early diagnosis of a tuberculous joint careful attention must be paid to detail. The essential points are chronicity, pain, the characteristic deformity, depending on the joints involved, limitation of movements of the joint, wasting of the surrounding muscles, and usually limitation to one articulation. This latter is by no means always true, especially in childhood, in which it is by no means infrequent to have more than one joint involved,—*e. g.*, the spine and hip, or both hips, or a hip and a knee involved together. In some cases a further test may be applied, which I will describe later. I refer to the injection of tuberculin as a diagnostic test.

Of late there has been an effort made by many to establish the fact that many cases of so-called chronic rheumatoid arthritis, or chronic polyarthritis, are really of a tuberculous nature. At first sight there does not seem much resemblance between the typical tuberculous joint, the outline of which I have just sketched, and the cases of chronic polyarthritis, the main characteristics of which are the involvement of many joints, and often the smaller joints which are not so commonly affected with tuberculosis, and the non-tendency to caseation and suppuration. This type of arthritis is quite different from the common and so well-known type of tuberculous arthritis. But the tubercle bacillus acts very differently in different cases. If we look for a moment at the method in which the lymphatic glands are involved we shall see what widely different clinical courses this disease may take. The usual type is familiar, the enlarged gland or glands ending in caseation and suppuration. But there is a form of chronic lymphatic tuberculosis in which many glands may become involved and in which there is no tendency to

softening and suppuration. Tuberculosis of joints may, therefore, from analogy, be expected to run varied clinical courses, the usual course being generally limitation to one or two joints, and if untreated eventuation in caseation and suppuration. On the other hand, a multiple chronic polyarthritis may be set up, showing no tendency to suppuration. The difference between these two forms of lymphatic tuberculosis is quite as striking as the difference between these two types of arthritis, and so what at first appears almost impossible may, by comparison with other organs, seem quite probable.

The main facts which have been brought forward in the attempt to show that some cases of chronic polyarthritis are really of a tuberculous nature are the following: The frequent development of pulmonary tuberculosis, or other form of clearly-recognizable tuberculosis, in the subjects of chronic polyarthritis; a number of cases, the subjects of chronic rheumatism, have clearly developed in one or more joints a condition that was clearly tuberculous; an injection of the fluid obtained by tapping such a joint has produced in a guinea-pig tuberculosis; and finally some observers have laid great stress upon the radiographic appearance of the bone-ends.

The subjects of chronic polyarthritis have sometimes upon them the evidence of tuberculosis, healed or active. Healed tuberculosis in the form of scars from lupus or cervical glands; active tuberculosis occasionally in the lungs or cervical glands. A family history of tuberculosis is not infrequently obtained. This may not be regarded as of much value. Personally I should place some importance upon it, when we are dealing with an obscure condition in which every little piece of evidence aids in the diagnosis. I admit it is likely to be treacherous at times.

Aspiration of fluid from these joints and inoculation experiments can only rarely be performed. The radiographic appearances are by no means confirmatory, but may at times be suggestive.

A test which may sometimes be employed in doubtful but suspicious cases is the injection of tuberculin. The value of this test has been very much debated, and there appear to be certain drawbacks to its use. There is said to be some danger, in the injection of tuberculin, of lighting up infection which has become quiescent. Some authorities however distinctly deny this. The test cannot be used in those pa-

tients who have some fever apart from the injection of tuberculin. This is, however, not very common in cases of chronic polyarthritis. The reaction is obtained not only in cases of active tuberculosis, but also in old quiescent cases, and in all persons suffering from undoubted tuberculosis the test is found not to be positive. The test has been found to be positive in persons not suffering from tuberculosis. We gather from this that there may be a small element of danger attending the injection of tuberculin, and it should never be employed merely out of curiosity. Secondly, the test is not infallible, and it is estimated that there is about 10 per cent. error in it. Nevertheless, I am sure that in some cases it will be found of use.

I can recall a patient, about thirty-two years of age, who for some months had been suffering from a swelling around her ankle. When examined, there was seen a swelling around the ankle-joint extending on to the dorsum of the foot, with a certain amount of pain and heat, and evidently of an inflammatory nature. I had very little doubt but that it was tubercular. An injection of tuberculin was administered, with a negative result. This was repeated, with a like result. A few weeks later the swelling entirely disappeared, without any treatment whatever. It probably therefore was not tubercular. Some few months later the swelling returned as before, and again the tuberculin test was negative. The nature of this must remain in doubt, but in all probability it was not tubercular, but more likely some septic condition.

By making use of this tuberculin test, some cases of chronic polyarthritis have come to be regarded as tubercular in nature. In that condition in children called acute rheumatoid arthritis (and as it was first described by Still is often called Still's disease), tubercle is regarded as the cause in many cases. Pulmonary tuberculosis has supervened in some cases, and the tuberculin test has been positive in other cases. Such cases are characterised by a more or less abrupt onset, with fever, a diagnosis of rheumatism being made. The symptoms abate somewhat, but the swollen joints remain, and vary considerably. The swelling is more around than in the joint. The lymphatic glands and spleen are enlarged also. In all such cases the tuberculin test has not been positive, but that it has in some is suggestive that at times these joints may be of a tubercular nature. In other cases the etiology may be different, and may probably be

some septic condition. It is quite a distinct disease from the chronic osteoarthritis of adults.

Syphilis of the joints has not yet received sufficient recognition, particularly in the case of children. It is a condition which has always been regarded as an extreme rarity, and whilst admitting that it is very rare, I do not consider it as rare as is supposed. Some cases are sometimes mistaken for tuberculosis and treated accordingly, much to the detriment of the joint concerned. Such treatment, if sufficiently prolonged, tends to end in ankylosis, and such patients may become permanently crippled, with a stiff joint or joints, whereas if treated primarily for syphilis the joint would prove to be very amenable.

The joints may be affected either in the inherited or acquired disease. It is more frequent, I think, in the inherited than in the acquired disease. It has been estimated that seven per cent. of all diseased joints in children are due to syphilis.

The two more frequent forms of joint disease encountered in congenital syphilis are, (1) the syphilitic epiphysitis of infants, and (2) the synovitis of older children.

The syphilitic epiphysitis, although not strictly a joint affection primarily, yet is so near the articulation and so often associated with some synovial effusion, and confused with joint disease, that it may be considered in this connection. This condition is more often met with in hospital than private practice, and from private case records no doubt it is a very infrequent affection.

In by far the majority of cases syphilitic epiphysitis occurs during the first few months of life. Nearly all cases are seen in the first six months, and about the third month is the commonest period. The oldest patient seen by Carpenter was seventeen months. Taylor says it may occur as late as the twelfth year. Such late cases are very rare. The infant may have presented other signs of syphilis previously, or have signs of syphilis upon it when the epiphysitis occurs, and in such cases the diagnosis is easy. But as often as not this epiphysitis is the sole manifestation of the disease, and such cases present considerable difficulties in diagnosis. Of the epiphyses involved the elbow, wrists, and knees are the more frequent, but all the epiphyses may be involved, even those of the ribs and clavicles. A ring or collar of enlargement is seen and felt at the

end of the bone, at the diaphyso-epiphyseal junction. This more or less completely surrounds the bone, is generally smooth and regular, and owing to some periosteal effusion this thickening often extends some little distance along the shaft of the bone. Some effusion generally occurs into the neighboring joint. One epiphysis only may be affected, or, as is more common, several; and the lesions are by no means infrequently symmetrical.

The two conditions most likely to be confounded with syphilitic epiphysitis are the enlargements of the bones which occur in rickets, and secondly, a septic infection of the epiphysis. The confusion with the rickety enlargement cannot often occur, but it sometimes will. If only one, or one or two epiphyses at the most, become involved, it is not rickets, which tends to involve many bones. It is in the cases in which many epiphyses are involved that the diagnosis is rendered difficult. In these the age of onset is the first important consideration. Rickets rarely manifests itself in the bones in the first few months of life, whereas syphilitic epiphysitis is nearly always seen at this early age. Syphilis tends to involve the larger epiphyses of the limbs, and the ribs are rarely affected, whereas in rickets the so-called rickety rosary is a very common, almost constant manifestation. Syphilis often tends to enlarge the shaft of the bone by periosteal implication extending from the epiphysis; in rickets the enlargement is restricted to the epiphysis. In syphilis some effusion into the joint generally occurs, but is absent in rickets. Rickety enlargements are not painful or tender; except in the acute cases, syphilitic inflammations generally are, and the latter are often associated with acute or subacute manifestations of inflammation of the skin around the epiphysis and joint. A history of syphilis in the child may be obtained, or other manifestations may be present. The latter are as often as not absent, but one feature I will call attention to in these syphilitic infants, viz., the puny, withered-up, old-age appearance of the infant. By attention to these points a correct diagnosis may be made and confirmed by the result of treatment.

When syphilitic epiphysitis originates acutely its likeness to a septic infection of the epiphysis or joint is very marked. The correct cause of the disease in such cases may be suspected by noting the comparative freedom from pain and tenderness, the absence of much constitutional disturbance, the fact that many epiphyses may be

involved, and the general aspect of the child with a syphilitic taint. An interesting example of this has recently come under my care:

An infant seven weeks old was admitted with a swelling around the vertebral border of the scapula. This was incised for diagnostic purposes and found to be solid. It was excised, microscopically examined, and diagnozed a fibrosarcoma. The infant developed a most extensive and severe erysipelas, from which it recovered and left the hospital. A week later it was brought back with a swelling around the knee-joint, which on examination was found to be a thickening around the upper tibial epiphysis and some effusion into the joint; the skin around was reddened and edematous. The pain and tenderness were very little marked, and constitutional disturbance was almost absent. As the child had had a recent attack of erysipelas and a suppurating wound, there was every reason for regarding this as a secondary septic epiphysitis. This was negatived by the comparative absence of pain in the swelling and constitutional disturbance, and a diagnosis of syphilis was made from the general aspect of the child. Syphilis had not been previously suspected. Mercury was given and a rapid improvement effected.

Syphilitic epiphysitis will sometimes cause a separation of the epiphysis from the diaphysis, which with an absence of previous history may be mistaken for a traumatic separation of the epiphysis. The latter, however, never occurs at this early age. A septic infection may occur secondarily in an epiphysis already affected with syphilis. Suppuration, necrosis, and even septic arthritis will follow. The original cause here is very likely to be overlooked, and yet it is most important that a correct diagnosis be made, since treatment, even with a separation of the epiphysis, will result in union. In all epiphyseal affections of infants one should bear in mind the possibility of syphilis as the prime cause of the affection.

The second most frequent affection of the joints in congenital syphilis is a synovitis, which occurs generally about puberty, or between the ages of eight and fifteen years. This is one of the more frequent manifestations of congenital syphilis which occur in older children, or about puberty. It is quite common to have no previous history of syphilis in these cases, although the physiognomy will sometimes reveal the evidence of the disease, in the shape of the nose or radiating scars from the angles of the mouth. In this form

of synovitis the larger joints are involved, and generally the knees. The tendency to symmetry is very marked. An effusion occurs into the joints. This is quite painless, and the only complaint is a feeling of weakness or insecurity in the joint, due to the general distension and relaxation of ligaments. Acute manifestations and suppuration are unknown. Such conditions are mistaken for slight sprains. The painlessness and symmetry should put us on our guard. Associated commonly with this are interstitial keratitis and deafness. An interstitial keratitis with deafness more or less sudden in onset, in the absence of middle ear disease, and a bilateral effusion into the knee-joints form an unmistakable clinical picture. Hutchinson's teeth and gummatous inflammation of the palate, leading to necrosis and perforation, may be present, as well as other syphilitic manifestations. These cases are unmistakable, but the joint affections may occur before these other signs, and it may be that by treatment the more severe and serious lesions may be averted. The essential points are the age of the patient, the painlessness of the affection, and the symmetry. These are suspicious. A careful inquiry into the history, and a careful examination of the patient for past evidences of nasal disease, mouth affections, bone lesions, and scars around the mouth may enable a correct diagnosis to be made.

In acquired syphilis the joints may be involved in the secondary or tertiary stage of the disease. In the secondary stage the type is one of synovitis with effusion. Sometimes a simple arthralgia occurs, and this quite early in the secondary period, even before other symptoms. The pain may be of some considerable severity. The synovitis of secondary syphilis has nothing distinctive in its nature. It occurs in the early period of constitutional syphilis. The diagnosis depends on the recognition of the primary infection and concomitant secondary signs, if present. Later in the secondary period the joints may again become affected, and it is generally the knee-joint which is thus involved. The characteristic here is effusion. Pain is often absent. This is by no means common in syphilis, but nevertheless will sometimes occur. The diagnosis depends upon the knowledge that syphilis is one of the causes of hydrarthrosis. Other signs of the disease may or may not be present. Even in the absence of other signs, a case of synovial effusion with no determining cause is always worthy of a trial of antisyphilitic remedies.

In tertiary syphilis a gummatous deposit is the chief characteristic. This may occur in the synovial membrane, either in an irregular manner, or it may be diffused more evenly over the whole membrane. Effusion may or may not be present. The similarity to tuberculous disease in these cases is very pronounced, and merely from the joint signs it is difficult or almost impossible to recognize the one disease from the other. The pain in a syphilitic joint is generally very slight, but, as mentioned before, this is sometimes the case in tuberculous arthritis. The limitation of movement is comparatively little marked in syphilis. But the main point in the diagnosis is the recognition of previous syphilitic infection and the reaction to antisyphilitic remedies. Such cases will generally speedily react to antisyphilitic measures, but it is a curious feature that relapses are quite common. If such joints are treated by fixation, as if tubercular in nature, the end will probably be some amount of ankylosis, but if recognized and treated for syphilis immediately perfect movement of the joint will result, and hence the importance of recognizing such cases. It seems not to be necessary to fix such a syphilitic joint; recovery will occur just as rapidly if free movement be allowed.

I can recall the case of a man, about forty years of age, who came complaining of a swelling of his right knee of some weeks' duration. On examining the joint the synovial membrane was thickened; the movements of the joint were only very slightly limited and pain was almost absent. I regarded the joint as tubercular, and was considering the best form of treatment to advise when the man developed an iritis. The correct diagnosis was thus given to me, and the joint rapidly subsided under antisyphilitic measures.

A more severe, and at the same time a more unusual, form of syphilitic arthritis is where the bone-ends, cartilages, and synovial membrane are all affected. The similarity to tuberculous disease and a nonsyphilitic osteoarthritis are in this case very marked. In syphilis it is said that the pain is much less severe than in the other diseases. The diagnosis can only be made in such cases by recognizing that syphilis will attack joints in this manner, by suspecting syphilis and endeavoring to obtain a history of the disease, and by noting the improvement from antisyphilitic remedies.

THE PREDISPOSITION TO, AND THE PREVENTION AND TREATMENT OF, PULMONARY TUBERCULOSIS

BY JOHN W. WAINWRIGHT, M.D.

of New York

The old and long-held theory of the hereditary transmission of tuberculosis, and the belief that a person is born with a tuberculous diathesis, are now scouted by many, if not by the majority of, medical authorities. The discovery by Koch of the particular organism which has been demonstrated to be the originating cause of pulmonary tuberculosis is mainly responsible for this change of view. The position of the greater number of scientific men as to the question of hereditary transmission of disease is somewhat as follows: While no disease of bacterial origin can as such be transmitted, and while no disease of quite extrinsic origin has been proved to be transmissible, there is a sufficiency of evidence to show that every malady due to an inborn vice of blood or innate taint of any kind may be transmitted from parent to offspring. Koch's bacillus then is the specific cause of tuberculosis, but predispositions to the disease either inherited or acquired are as essential to the development of phthisis as the bacillus itself. The germ of consumption is the seed, and the predispositions to the disease are the soil. Without a soil fitted for its reception the germ cannot gain an entrance and flourish, and the more suitable the soil is the more luxuriantly will the seed flourish. Lucas, in the Wightman lecture, 1904, touches upon the various theories of heredity, the "pangenesis" of Darwin, the "physiological units" of Spencer and the "ancestral germ plasm" of Weismann. He takes it that the theory of the hereditary transmission of tuberculosis is generally discredited. It is instructive to compare the opinions which prevailed fifty years ago with those of the present day on this subject. In 1840 Watson thought that consumption was entirely a matter of diathesis, and nothing was heard of the contagion theory of the disease. Even a wife whose family had been free from consumption for generations, who contracted phthisis

after nursing a husband dying of the disease, only proved to Watson how long may be the latent period in the generation before the taint declares itself. Lucas is of the opinion that all the old views relating to the tuberculous types of patients are not quite valueless. He holds that the pearly sclerotics, the sharp refined features that may be seen in any wasting disease, may be produced by the tuberculous process. This may be transmitted to the offspring, so that in the few generations who had suffered from the disease we would have the type occurring in a healthy individual, and merely indicating a diminution of resisting power. Lucas concludes by saying: "Physical deterioration rests on two factors; the defective health of the parents and the unhealthy surroundings and deficient food of the offspring. Of these two factors the parents exert the greater influence, because in them is summed up not only the result of their own unhealthy lives but that of their ancestors. Much, no doubt, can be done for the child by good food, good air, and mental and moral training, but he must carry the tyranny of his type to the end of his days. That certain diseases are to some extent hereditary would seem probable, but in the main the theory of the direct hereditary transmission of disease has fallen altogether into disrepute, although it may be that, as so often happens when old views are overturned, we rush too far to the other extreme.

Huber, in a paper read in October, 1903, on "Predispositions to Tuberculosis," considers both the prenatal influence and postnatal predispositions. Referring to prenatal influence, he says: "It is not merely a fancy of the romancer, but a statement saturated with practical sense, that the past lies upon the present like a dead giant's body, so that it is as if a young giant were compelled to waste all his strength in carrying about the corpse of an old, an atavistic giant. We sleep in dead men's houses, bacillus-ridden as they are; we are sick of dead men's diseases; we live in dead men's lives, as these sentiments are taken from a dead man's book. And it is equally true that future generations must inevitably be affected by the disposition, physical or otherwise, of the lives of individuals in the present." Huber points out that the opinions as to the heredity of tuberculosis are many and diverse, ranging from that of the transmission of the disease itself, to the declaration that tuberculous parents actually confer upon their offspring an immunity to

the disease. The larger number of medical men follow the teachings of Cornet, who holds that tuberculosis is a nonhereditary disease, and that the child begotten of parents, one or both of whom are tuberculous, is only predisposed to tuberculous infection. Conheim was probably the first to suggest the possibility of a direct transmission of tuberculosis to the embryo. Baumgarten accounts for the occurrence of infantile tuberculosis by assuming that the bacillus penetrates the sperm cell or ovum and remains latent for some years, quoting in support of this view the analagous instance of pébrine, in which Pasteur worked out the mode of infection. In considering this theory the fact must be borne in mind that specific congenital infection by the Koch bacillus has been stated, on the authority of several investigators, to exist.

Huber, before reviewing postnatal predispositions, draws attention to certain aspects of struma—the pronounced sluggish metabolism, and the tendency to inflammation of mucous membranes and of lymph-glands, the respiratory membranes and the glands of the mesentery and the bronchi being marked sufferers. Flick, Wright, Dickinson and others tell us that in the beginning tuberculosis is a purely local affection. Tissues which have had their circulation disturbed by traumatism, vasomotor, functional, or other disorders, become foci. The extension of the process then depends largely upon the spread of the bacillus from these diseased foci by means of the lymphatics and the blood-vessels. Inhalation of the Koch bacillus into the air vesicles as the primary cause of pulmonary tuberculosis is considered by many a wrong view. It is held that the bacillus cannot be inhaled because the function of the respiratory tract anatomically and physiologically is limited to the admission of gaseous substances only. The main routes by which the lung tissues are invaded are then two: First, by way of the alimentary canal, the course being by the stomach, intestines, lacteals, thoracic duct, the vena cava, the right heart, and the lung; the second route being by way of the bronchial glands to the thoracic duct, the vena cava, the right heart, and finally the lung. There is no doubt whatever that the alimentary tract is very intimately concerned in the spread of tuberculosis. According to Sájous, vulnerability to tuberculous infection means insufficiency of the adrenal system and lowered oxidation. It is obvious that a faulty metabolism plays a

most important rôle in predisposing the tissues to tuberculosis. It also goes without saying that an impaired circulation is a predisposing factor of great moment in the causation of tuberculosis.

Chlorosis is frequently related to tuberculosis, and to whatever cause the condition may be ascribed its relation can be logically explained. Magnus Huss in 1849 originated the belief that the sclerotic action of alcohol was antagonistic to phthisis, but now the opinion that Laverenne promulgated in 1780, that alcohol is one of the predisposing causes of the disease, is almost universally accepted. Alcohol taken in excess renders the body less resistant to diseases of all kinds. The expression of the views of Professor Sims Woodhead in England and of Laitenen in Germany tend to support this position. Osler has stated that chronic drinkers are much more subject to both acute and pulmonary tuberculosis. It is probably, he thinks, altogether a question of altered tissue soil, alcohol lowering the vitality, and enabling the bacilli more readily to develop and grow.

The French, however, are the greatest exponents of the view that alcohol is one of the most fertile predisposing causes of tuberculosis. Professor Brouardel said: "Alcoholism is the most potent factor in propagating tuberculosis. The strongest man who has once taken to drink is powerless against it." The opinions of the French medical profession generally on the matter are well summed up in the aphorism of Laudonzy: "L'alcoolisme fait le lit de la tuberculose." Indirectly, the fact is patent that alcoholism is a fruitful cause of consumption, by lowering the vitality of its victims, who usually either drink because they are poor or are poor because they drink, who live in unhealthy surroundings, and eventually become themselves degenerate, begetting degenerate children, susceptible in a high degree to disease. Poverty, with its associations, is so obvious a predisposing cause that it needs little mention. Insanity and tuberculosis have a connection, which, however, it is not easy to trace. Altitude, temperature, moisture, season, and soil may be predisposing factors. Tuberculosis often follows such diseases as bronchitis, influenza, and neglected colds, while convalescents from the exanthemata, diphtheria, and typhoid are subject to infection by the tubercle bacilli, and the coexistence of maladies like syphilis, malaria, and diabetes are powerful elements in the growth of tuberculosis. Many

trades predispose to tuberculosis, as do various forms of direct injury, but Huber thinks that traumatism often results in tuberculosis by exciting nervous reactions. In this connection Huber points out that the influence of the mind upon the body is a factor to be considered in the development of phthisis.

TREATMENT

With regard to the treatment of consumption, we stand on much firmer ground. Medical men in all parts of the world are now agreed as to the broad lines of treating pulmonary tuberculosis. It is now known that all children, with but few exceptions, can be saved from the disease if they are sufficiently defended from the inhalation or ingestion of the fungus-like germs. The *Edinburgh Review,* October, 1901, points out that a predisposition is inherited, and so long as our streets and railway-carriages and infected houses are allowed to collect sputum dust, predisposed persons run a risk in frequenting such places, but when law and habit combine in precautionary measures, especially in restraining expectoration, these will, in spite of a susceptibility either constitutional or temporary, be nearly as safe against phthisis as against leprosy. We have as a result of knowledge of the causes of the malady and of minute long-continued investigation of the actual effects upon the patient of different modes of treatment, the most encouraging expectation of cure in the majority of cases of which the character has been ascertained at an early stage.

It is only within quite recent times that the rational treatment of phthisis, by fresh air and diet, has been carried out. At one time —and not long ago—a consumptive was wrapped up, metaphorically speaking, in cotton wool, and guarded from the air, and especially the night air, as if it were the plague. Although many years ago some men raised their voices in expostulation at this coddling mode of treatment, they were unheeded. The late Sir William MacCormac's father, Dr. MacCormac, of Belfast, was one of the first to advocate open-air treatment. Now it may be stated in a broad way that it has been indubitably proved that with men, as with animals, phthisis does not occur in out-door life, where it has not been introduced from a previous case, while it occurs very rarely among people who avoid crowded places or vehicles, who live much in the

open air, and whose houses are roomy and clean. The converse of this statement is also true.

The treatment of phthisis then in these days is by means of fresh air, sunlight, and an appropriate and generous diet. Even sleeping out in the open air has been attended with good results, and is preferable to passing the night in a small room with the window open. Millet, of Brockton, Mass., reports the cases of five patients, whom he recommended to sleep out of doors at night. The outcome in each instance was most satisfactory.

Sanatorium treatment of consumption has been most successful. To the Germans is due the credit of initiating this mode of treatment, and it is in Germany that sanatoria are most numerous and perhaps most efficient. According to Latham the essentials of sanatorium treatment are:

(1) A continuous supply of fresh air with no unnatural changes of temperature and avoidance of all sources of irritation from dust.

(2) Good, nourishing food in sufficient quantity to maintain the normal body of the patient.

(3) An absolutely regular life, so arranged that neither the lungs nor any other portion of the body is under any strain, and

(4) Graduated exercise without strain.

Fisher describes sanatorium treatment somewhat as follows:

In fine weather the patient can pass the day out of doors with adequate shelter from wind or hot sun. On wet or stormy days he may rest in a suitable shelter or piazza. In the larger number of sanatoria huts are erected in different positions, those for winter facing south and others for summer facing north. Balconies with proper protection from wind, rain or glare are suitable for weaker patients. At night the head of the bed should be close to the open window. Shelter from strong winds or rain can be secured by moving the bed about, or by some screen arrangement. The larger the window space the better, since the air of the room is more easily kept pure, and there are fewer spaces for stagnant air. It has been found that febrile patients do better in a châlet with windows on all sides than in an ordinary bedroom with open windows. The furniture should be scanty; carpets, curtains, wall papers, and everything that can harbor dust should be abolished. Hence the walls should be smooth varnished or distempered; the utmost cleanliness is es-

sential, and proper precautions should be taken regarding sputum and disinfection. If possible, every patient should have a separate bedroom, to prevent disturbance by talking, coughing, etc., and to ensure privacy and rest. The site of a sanatorium should be especially chosen to afford pure air, with shelter from strong or cold winds. Therefore it should be quite in the country, away from smoke, dust, fog or noise; isolated in its own grounds.

The majority of consumptives do best living at an altitude of some hundreds of feet above sea level, but many patients improve greatly in low, sheltered localities. Nevertheless, it is well that the place be not so surrounded by trees and vegetation as to prevent free circulation of air. Proximity to the sea is not generally advised for consumptives. According to Mangnat, the tuberculous may be classified in four categories:

(*a*) Torpid tuberculosis in persons whose nutritive exchanges are normal or subnormal, whose circulation is calm, whose lesions are circumscribed and stationary. These may improve at the seaside.

(*b*) Those whose lesions evolute per saltum—*i. e.*, acute outbreaks followed by periods of remission of variable duration. These should be sent to a sedative station, and residence too near the sea must be prohibited.

(*c*) Cases of acute, rapidly-progressing tuberculosis should never be allowed near the sea.

(*d*) The incurable cases, in which the choice of a climate is of less importance and may be left in great measure to individual taste.

As to the climate most suitable to consumptives, there is less difference of opinion than was formerly the case. It used to be thought that a variable or moist climate was most harmful to the consumptive. At the present time the question has been relegated to the background, as it has been found that frequently patients do very well in moist and even bleak climates. Idiosyncrasy is a factor in the matter; a place which suits one does not suit another, but as a practical rule, the consumptive is best treated in the country in which he has to live.

Rest is an essential feature of the sanatorium treatment. Various conditions, such as hemorrhage, high temperature, debility, etc., indicate complete rest in a recumbent position. It is a good plan

for patients to rest an hour before the principal meals, talking also being discouraged. Thus the patient comes into his meals with quiet pulse and breathing, and digestion is undisturbed. At some sanatoriums constant rest is prescribed, but this is not now the rule, except for certain cases.

That graduated exercise is an important factor in the sanatorium treatment is now almost unanimously allowed. Brehmer was the first to insist on this, and his views have borne the test of trial. Injudicious exercise, however, is to be greatly deprecated. Exercise should be given gradually and tentatively. The effect upon temperature and pulse should be noted. Even when the condition is febrile, some patients do not improve until gentle exercise is taken. Walking is the most usual mode of exercise, the pace being regular and slow. As strength increases, so should the walks be longer. The extent and direction of the walks is under medical supervision, and for weakly patients sheltered walks are chosen. Rests should be frequent and patients are not to be sent out in groups. The stronger patients should not be deterred by inclement weather, but resting in wet clothes or upon damp grass or seats is contraindicated. After a time hill-climbing may be encouraged, and some simple form of drill, with breathing exercises, is beneficial when the disease is quiescent. Patients should walk with an upright carriage.

The diet of consumptives is a rather vexed question, not indeed as to the quality, but the quantity thereof. The German sanatoria as a rule are accustomed to cramming the patients with food, making them take a certain very large amount during each twenty-four hours. The American and English sanatorium physicians, on the contrary, in a general way feed generously, but not excessively. Galbraith is of the opinion, from a histologic point of view, that rational dieting consists, not in indiscriminate stuffing, nor in indiscriminate lessening of the food, but in furnishing a diet rich in the appropriate albuminous constituents. The diet in consumption is to be regarded as possessing a directly curative value, and its chemical composition is of as paramount importance as the constitution of the active principles of any mixture, however indifferent may be that of the vehicle. The special advantage of a meat diet in this relation is that, besides containing the nuclear bodies which have a directly stimulant action, it contains also proteids which have a high nutrient

value. Sufficient fat and carbohydrate must be given to meet the physiologic needs; the amount may be increased later, as metabolism improves to the maximum. Goodbody, Bardswell, and Chapman as the result of investigations on metabolism in phthisis conclude:

(1) Both clinically and experimentally tuberculous patients show very satisfactory results on their diet being increased slightly above the original, and it is evident that the state of the appetite gives too low an estimate of the requirements.

(2) Comparatively large diets are well borne by those who are much below their normal weights, but not so well supported by patients up to their normal weight and with arrested disease.

(3) In all cases very large diets give unsatisfactory results, as the gain in weight is at the expense of the general health, as indicated by the failure of appetite, marked digestive disturbance, and increased intestinal putrefaction.

(4) Moderately large diets give the best results.

(5) The assimilation and absorption of nitrogen is good, on the whole, even in cases of high pyrexia. Absorption of fat is excellent.

(6) Patients complain least of discomfort on diets which give the best results experimentally, and the onset of severe dyspeptic trouble coincides with deterioration in the experimental results.

(7) In patients in which the disease is becoming quiescent and the patient is approaching his normal weight it is inadvisable to diminish the quantity of food; but even at this period a more substantial diet is necessary than in health.

Patients with active disease and pyrexia require careful and systematic dieting. In an ordinary way three good meals are given a day, with a half to a whole pint of milk at each. Milk should also be given at stated intervals. Fats, such as butter, cream and honey, should form a part of the diet.

Medical supervision is generally allowed to be a main factor in treatment. In the words of Latham: "The physician must be an autocrat; his word must be law; he must be prepared to give his entire energy to the work, for, as Dettweiler has expressed it, 'A sanatorium must be his religion, his politics, his despair, and his delight.'"

With regard to the duration of treatment, Fisher says: "The

usual time of treatment is from three to six months, sometimes longer. It may in some cases take two years or even longer." The majority of sanatoria only admit those suffering from tuberculosis in its early stage.

There are many sanatoria in all civilized countries for patients who can afford to pay, but few for those who cannot. The truth is that sanatoria are far more needed for the latter class than for the former, as the poor are the more potent means of spreading the disease. Otis thinks that for the treatment of the consumptive poor two sets of institutions are required,—hospitals for consumptives in or near the city, for the reception of poor consumptives in all stages of the disease, erected and maintained by the municipality, and several state sanatoria favorably situated climatically and not too far from the city, which would furnish the majority of its clientèle.

Within the past few years camp life has greatly gained in favor as a means of treating phthisis. Reports from all parts of the United States where tent life for tuberculosis has been tried, are favorable. Stone recommends it, as does Craig, while Stubbert is convinced of the remedial and curative effects of fresh air in cases of phthisis. Waxham, however, points out that tent life should be under medical supervision, and that, in addition to the air, the patient should have food, rest, and comfort; that the strenuous life of a healthy camper should form no part of the invalid life. There are those who hold that a cabin is preferable to a tent for the accommodation of the consumptive, as a cabin is more easily effectively ventilated. But a tent, if well selected, will answer the purpose well. A wall-tent is perhaps the ideal arrangement for the summer, the sides of which can be raised day and night and only lowered to protect the occupant from strong winds or driving rain. For the winter in a cold region a more substantial tent is necessary, of which the ventilation is perfect, and which can be heated to 60° or 65° Fahrenheit when needful. Dr. A. E. Macdonald, Superintendent of the Manhattan State Hospital, East, Ward's Island, New York City, inaugurated a system of tent care for the phthisical insane which has been attended with satisfactory results.

In order to cope with consumption with hope of ultimate success, the treatment of the poor in large cities is the problem which must be solved. Sanatoria for those who cannot afford to pay are

absolutely necessary, and must be built on a large scale. This, however, is a matter which rests chiefly with state legislatures and municipalities.

To the great majority of poor consumptives the only available treatment is the home treatment. There are many who, if there were institutions to which they could go, are unable to leave their homes. In the city of New York there is accommodation for about four thousand of her consumptive poor, while there is requirement for ten to twenty thousand more. Thus home treatment is an essential measure to be considered in the fight against phthisis.

While the treatment of consumption is of the utmost importance, its prevention is even of greater importance. As long as the poor of great cities live huddled together like animals, in unsanitary, badly-ventilated dwellings, just so long will the disease originate and spread. The most efficacious single reform for the prevention of pulmonary tuberculosis will be the admission to every dwelling of the largest possible inflow from the vital atmosphere.

Public buildings, schools, theaters, and churches, by reason of insufficient ventilation, afford, frequently, a potent means of originating and spreading pulmonary tuberculosis. In railway cars, on steamships with close, stuffy cabins into which a constant succession of infected and healthy individuals are admitted with no due precautions, the seeds of consumption find a favorable soil for propagating and flourishing. The house, however, may be termed the granary of the tubercle bacillus outside its host. Flick avers that were it not for the house the tubercle bacillus would soon have to perish from the face of the earth. It could not be preserved. Sunlight, air, and water are its natural enemies. As to how long tuberculous matter will keep alive in the open air has not been determined with exactitude, but it is agreed on all sides that the time is short. Under such conditions when the bacilli are not destroyed their virulence is so weakened that they will not readily take root in the human system. In the house, on the other hand, tuberculous matter will remain vital for a considerable period, because it becomes dry and the broken-down tissue serves as a nidus for the germs. So long as the sun and air are prevented from exercising their influence upon the bacilli, these will continue to be a source of danger.

Hoffman concludes that the decrease in the prevalence of phthisis is due to ten factors, and these cover the whole subject in so illuminating a manner that they are worthy of quotation:

(1) We may attribute the decrease in mortality from tuberculosis to vast and far-reaching improvements in the housing of the masses of the population in cities, of which formerly a large proportion lived in cellar dwellings, in houses located on damp and unwholesome subsoil, or on areas filled in with garbage and other health-destructive matter.

(2) There has been a vast improvement in the direction of providing tenements and dwellings with more light and better ventilation, and prohibiting entirely the use of dark interior and ill-ventilated rooms for living and sleeping purposes.

(3) The evils of overcrowding, while still considerable, are without question less serious at the present time than formerly.

(4) Intemperance, while still an evil of great magnitude, is now less of a bodily destructive character than fifty, or even thirty years ago.

(5) There has been considerable improvement in the feeding of infants and adults with food of a more nutritious quality. Also, the more vigorous health, especially among women, is, in a large measure, due to the greater amount of outdoor life, and also to the public parks and children's playgrounds, which a more enlightened public opinion demands.

(6) Improved economic conditions, especially in the direction of higher wages and shorter hours of labor, and doing away with night labor, has resulted in a more healthy development of body and a higher degree of disease resistance.

(7) Child labor is now practically prohibited and made impossible by law in most of the states.

(8) Unwholesome and unsanitary conditions of factory life, of serious effect on health and longevity; dust-producing occupations, especially destructive to lung tissue, and numerous other evil conditions surrounding men and women employed in ill-ventilated factories and workshops, have been largely done away with in the light of modern sanitary science, through factory and labor legislation.

(9) School life, or the conditions surrounding children at the impressionable age of the educational period, have been materially

improved by slow degrees from the close, overheated school-houses of the past to properly lighted, sufficiently roomy and well-ventilated school-houses of the present.

(10) The general social improvement of the mass of our population, making for a higher standard of life and the worth and value of living.

The foregoing, then, have been the means in those cities in which they have been carried out in decreasing the prevalence of phthisis. Much, however, yet remains to be done. In many cities hardly a beginning has been made, but sufficient has been demonstrated in a few to show clearly that such are the principles upon which we must work in order successfully to fight consumption.

It may then be affirmed with more or less certainty that nearly all the phthisis of adults has been forced as in a hothouse, by overcrowded and stuffy homes, schools, public buildings, workshops and railway-cars, and spreads thence to people in better conditions by habits which ought to be tabooed as both disgusting and dangerous. These objectionable habits will gradually disappear under the influence of public opinion and thorough municipal regulation; but of course these manifold improvements will take time and it will not be for many years that their full effect will be seen upon the public health.

Tibbles mentions, among other preventive measures, that tuberculous mothers should not suckle their young; nor should their children be brought up by hand, if this can be avoided—the best means of fitting the infant to resist the disease in its many forms is by providing it with a good wet-nurse; that phthisical people, and those of strong phthisical tendencies, should not marry; and that phthisical persons should not occupy the same bed or bedroom with a healthy person.

By carefully regulating preventive measures the spread of phthisis can be stayed, and by rational means of treatment, by fresh air, sunlight and diet, the disease can be arrested and cured in its early stages.

Dr. Brouardel, the distinguished French physician, goes so far as to say that phthisis is avoidable and curable and that we may reasonably prophesy that united in one brave attempt the whole civilized world will succeed in exterminating this, the cruelest scourge.

MILK AND MEATS IN THEIR RELATION TO THE SPREAD OF PULMONARY TUBERCULOSIS

Koch's statement at the British Congress of Tuberculosis in 1901, that human and bovine tuberculosis were absolutely non-identical, and that consequently infected milk or meat had nothing to do with the origination or spread of the disease, has caused the whole matter to be closely investigated. Virchow was sceptical as to the validity of Koch's reasoning. Hueppe was also a doubter, while Behring has asserted that Koch was misled.

Adami, Ravenel, Salmon, Dèlepine and many others have published within the past few months the result of investigations going to show that infected milk at least is a means of conveying tuberculosis to human beings and especially to infants. Ravenel has made long-continued researches into the matter, and thinks that the subject may be approached from three standpoints,—first, direct experimental transmission from man to animals; second, clinical evidence of its transmission from animals to man; and third, theoretical considerations.

The American, British and French bacteriologists, as a rule, hold the view that bovine tuberculosis is a factor in the causation of human tuberculosis. Chauveau was the first, in 1868, to prove that cattle could be infected with tuberculous material from man. In 1879 Bollinger succeeded in similar experiments, while Klebs, Crookshank, Kilt, Martin, Thomasen, Nocard, DeLong, Westenhoffer, Wolff, Schottelius, point to the fact that Koch was wrong, or, at any rate, his pronouncement was altogether too dogmatic.

The first point is susceptible of direct experimental proof. Febiger and Jensen succeeded in transmitting human tuberculosis to cattle. Young and Hamilton published, in the beginning of 1904, the list of experiments undertaken by them. They began with 20 calves, one of which died during the preliminary period of observation. Nineteen were inoculated with human material, and of these fifteen developed tuberculosis, while four resisted the inoculation.

The next part of the subject to be considered is the question of transmission of tuberculosis from cattle to man, and in this connection there is no direct experimental evidence to go by. However, a number of cases have occurred in which accidental inoculation has taken place, some of which have proved fatal. Cases of this de-

scription have been reported by Krause, Troje, Spronk and Hofnagle, Lassar, Tschering, Pfeiffer, Hartzell, DeLong, Joseph and Trautman. This phase of the question must be considered under the head of ingestion. Experiments have shown that animals can easily be infected by ingestion, and there is sufficient evidence also to demonstrate the occurrence of human infection by this means. Of the cases of clinical evidence through feeding which have been published may be mentioned those of von Ruck, Stang, Demme, Gosse, Ollivier, Law, Ebers, Bang and Rievel. In these instances the evidence is very direct and strong. In line with infection by ingestion post-mortem evidence has to be considered. Ravenel points out that this kind of evidence is somewhat contradictory. In Great Britain there is a general average of primary intestinal tuberculosis of over 25 per cent. Guthrie reports, at the Children's Hospital at Paddington, 24.6 per cent.; Still, at the Great Ormond Street Hospital, 23.4 per cent.; Shennan, at the Royal Hospital in Edinburgh, 26.1 per cent. In Germany, on the contrary, the average is low, although Professor Heuppe states that the percentage of primary intestinal tuberculosis in Germany varies from 25 to 30 per cent.

In America the collection of figures on this matter is comparatively small, but they tend to show that primary intestinal infection is of somewhat rare occurrence.

The strongest evidence available of the infection of human beings by the bovine tubercle bacillus is the finding of these bacilli in the intestines of children who have died of intestinal tuberculosis. A case of this description was reported from the laboratory of the State Live Stock Sanitary Board, sent by Dr. Alfred H. Hand, of the Children's Hospital in Philadelphia. Theobald Smith found a similar case, and De Schweinitz, of the Bureau of Animal Industry, found two cases. Some cases have also been reported from Europe.

The bovine tubercle bacillus has been shown by whatever method of inoculation may be used to be far more virulent than the human bacillus. The first report of the German Commission on Tuberculosis was issued in the summer of 1904. The conclusions arrived at so far are as follows: Taking for granted that a "perlsucht" infection has occurred in man, if bacilli, indistinguishable from those of "perlsucht," can be separated, the commission conclude that out of three cases in which the primary foci were found in the ali-

mentary system, one was of that origin. Adding to these, seven cases of primary tuberculosis of the intestine or mesenteric glands in which two had arisen from " perlsucht " infections.

The Commission believe with Koch that human and bovine bacilli are of different species. It must be borne in mind that Ravenel and others have shown that the virulence of human bacilli can be greatly increased by passing through a series of animals of the same species, so as to approximate in pathogenic power to bovine bacilli. The first findings of the British Commission on Tuberculosis, also published in 1904, tend to show that human and bovine tuberculosis are identical, so that the matter is in an undecided state. On the whole, perhaps, the majority of American physicians will be inclined to agree with Ravenel, who says: " We are then justified in saying that bovine tuberculosis is transmitted to the human race, chiefly to infants, in a certain proportion of instances, and is, therefore, a menace to human health. Our present knowledge does not enable us to define exactly the extent of the danger, but that it exists cannot be denied. It is, therefore, the duty of physicians and officers of health to take every precaution against the infection of many by tuberculous cattle and their products."

SUPERVISION OF MILK SUPPLY

Even if it cannot be absolutely proved that bovine tuberculosis can be transmitted to man, enough has been done to demonstrate that such is probably the case. Consequently, every possible care should be taken to ensure an uncontaminated milk supply. Behring contends that milk is the main source of infection in human beings, whether it be the milk of the tuberculous mother or that of a tuberculous cow. He makes the following distinction between infants and adults in their liability to infection through the digestive tract. In the young of man and of many other animals the intestinal epithelium is so constituted as to permit the entrance into the blood-stream of bodies which the mucous membrane of the adult excludes. The pores of the intestinal filter in infants are large and readily permit of the passage of various microörganisms, tubercle bacilli among others, whereas in the adult these pores are much finer. Romer, for example, has shown that in the new-born, albuminous bodies can enter the blood unchanged; on the other hand, in adults they must

be split up by digestion into peptones before they can pass through the dialyzing membrane which in the developed individual constitutes the lining of the intestinal canal. For this reason, adults may drink with impunity the milk of tuberculous cows, infection through this means occurring only when there is some lesion of the intestinal mucous membrane, breaking down the normal defences.

The principal food of infants being milk, added to the fact that such vast numbers of young children are fed nowadays artificially, it follows that it is of the most vital importance, in the preservation of the race from tuberculous infection, that the milk supply should be as pure as possible. The milk supply of cities is on the whole by no means uncontaminated, and consequently milk is a fruitful agency in the conveyance of phthisis to the human race. Again, it is now generally admitted that milk unsterilized and unpasteurized is, when pure, a more nourishing food for infants than the product after it has been submitted to these methods. What steps then must be taken to ensure a pure milk supply in cities? In the first instance, the cows from which the milk comes should be healthy and free from tuberculous taint. It is notorious that there is a very large number of herds which are not free from this taint, and further, cows of high breeding are usually affected by tuberculosis to a greater or lesser extent. Thus the question of a high standard of milk is also a question of different breeds of cattle. The Holsteins and Ayrshires, the greatest milkers known, are comparatively free from tuberculosis, while the aristocratic breeds like the Guernseys and Jerseys, although they give a far richer milk, are almost invariably predisposed to tuberculosis. Of course, to stamp out tuberculosis in dairy herds thoroughly and effectually, all cows found affected with tuberculosis should be destroyed. Such a plan would be attended with so many and so great difficulties as to be almost impracticable. There is so immense a sum of money invested in the dairy business, that to order the destruction of all herds affected by tuberculosis would mean the loss of an enormous amount, part of which the state would have to bear. This situation has kept the subject in the background. If, then, these radical measures cannot be carried out, at least every possible precautionary measure should be taken to preserve milk from tuberculous infection. Cows proved

affected by tuberculosis should be destroyed and regulations enforcing cleanliness in all the arrangements pertaining to milking, transportation, and care of the milk in city dairies, should be most rigidly adhered to. Generally the tuberculous lesions in cows are restricted to the bronchial and throat glands, but occasionally the udders have been found to be affected. It must, however, be borne in mind that it is not necessary that the udders be diseased for the tubercle bacilli to gain entrance into the milk, and, therefore, it is essential to use all efforts to secure a milk free from such germs. So far as the question of eradicating tuberculosis from dairy herds is concerned, as mentioned before, it is impossible except by destroying all tuberculous cows; much, however, may be done in this direction by keeping cow stables in a healthy and clean condition.

Pierson, of Philadelphia, undertook an interesting experiment in this connection. He fitted up two stables in the University Veterinary School to show the contagiousness of tuberculosis in cattle under different conditions. In one stable he placed six cows, two of which had tuberculosis and four of which were healthy. This stable had only three thousand cubic feet of air-space and a few windows, and was in every way unhygienic. The other stable had nine thousand cubic feet of air-space and plenty of light, and was kept scrupulously clean, and in it were placed the same number of cows, two of which had tuberculosis and four were healthy. He found after seven months that the four healthy animals put in the dirty stable all had tuberculosis in a fairly advanced stage, whereas in the clean stable only two of the cows showed any tuberculosis, and that in a very mild degree.

Moore, referring to the milk question, summarizes as follows:

(1) Milk as it is drawn from the udder contains a variable, but rarely an excessive, number of bacteria.

(2) Ordinarily the dirt and finer particles of dust that fall into the pail during the process of milking carry a large number of bacteria with them into the milk.

(3) If the stables are kept clean, the cows groomed, and the skin of the udder and surrounding parts are carefully moistened before milking, the number of bacteria that gain entrance to the milk from without can be very greatly reduced.

(4) The bacteria in the freshly-drawn milk do not begin to mul-

tiply to any great extent for from six to nine hours when kept at a temperature of 70° F., or below. After that time they multiply very rapidly if the temperature is favorable.

(5) If the milk is carefully protected and promptly cooled down to and kept at a temperature of 40° F., the number of bacteria in it will not subsequently exceed the number present at the close of the milking process. This number should not exceed a maximum of 100,000 per cubic centimeter. If intelligent care is exercised, a minimum of at least from 10,000 to 50,000 per cubic centimeter should be maintained. If cooled to 55° F., like results are obtained for at least thirty-six to forty-eight hours, and often for a much longer period.

(6) An excessive number of bacteria in milk at any time within seventy-two hours after it is drawn indicates that it has not been properly handled.

(7) There is a pronounced germicidal action in the milk of certain cows. It is absent in that from others. In the mixed milk from a dairy it has but slight value in reducing the actual number of bacteria, but it seems to be the effective agent in preventing the increase of bacteria immediately after the milk is drawn, unless it is kept at the body temperature.

With many physicians both sterilizing and pasteurizing of milk has fallen into disrepute, and the movement against these methods seems likely to spread. Therefore, it becomes more and more incumbent on municipalities to ensure a pure milk supply, especially if tuberculosis be widely spread by this means. This object may be effected by legal regulations, by public opinion, by the example of high-class dairies, and by endorsement by responsible persons.

ON MILKY URINE

BY F. X. GOURAND, M.D.
of Paris

It is not unusual for a patient to come with troubled countenance to his physician and say that he is very uneasy because he has observed that of late his urine has no longer been clear, but whitish, somewhat like milk. For this symptom is one that alarms the public very much! And yet it is a matter which, though worthy of serious consideration, is by no means as grave as is usually thought; in fact, it may call attention to conditions that can be successfully treated.

Let us first see what is meant by milky urine, and distinguish it from other modifications of the urine for which it may be mistaken. The condition called milky urine is characterized by the quite peculiar aspect of the urine *at the moment of its emission.* It is uniformly non-transparent; of a more or less yellowish white, according to the amount of precipitate it contains; and it decomposes with great promptitude and its surface is then covered with a fatty and iridescent pellicle. When set to one side a white powdery deposit falls to the bottom, though the urine *rarely clears up entirely,* at any rate not until it has stood for a very long time.

Such are its principal characteristics, which are more or less marked according to the intensity of the condition. All the degrees between normal urine and marked milky urine are met with, though it must be said that even in the most pronounced cases the urine is never as opaque and white as milk; it resembles the latter, and nothing more. One only of the above characteristics seems indispensable: the urine must be non-transparent as it passes through the urethra. In this way milky urine will not be confused with urine that has undergone ammoniacal fermentation, or with a precipitate of urates through cooling off of the urine when it is concentrated. In certain cases of bladder infection ammoniacal fermentation takes place in the bladder itself and the urine passed is non-transparent;

but the general symptom-complex is then quite different, these cases being of a surgical order. There is one more source of error to be noted, though it can be easily avoided: purulent urine is also non-transparent as it passes out; but its greyer color, and especially its smell, its ammoniacal reaction, and above all a microscopic examination, will clear up all doubts as to the diagnosis.

It is unusual that all the urine passed during the day is milky; as a general thing, certain emissions only present this appearance. Thus, in many cases only the urine passed after meals is non-transparent; with other patients the milky condition lasts three or four days and then disappears, to reappear a few weeks later. There are few other symptoms connected with this condition; as a rule the patient only becomes aware of it by seeing the appearance of the urine. Still the emission may be slightly disagreeable or burning; in rare cases it may be accompanied by an attack of severe pain radiating along the urethra and toward the anus. In the latter instances the stream may be suddenly interrupted, and there may even be slight hemorrhages; for these really painful forms are due to the presence of phosphatic calculi which irritate the mucous membrane and sometimes make it bleed.

There is an entire category of patients, those with neurasthenia, with whom the emission of milky urine coincides with a recrudescence of their neurasthenic symptoms: headache, lassitude and lumbar pain; the pain is present of course all the time, but is often worse at the moment of passing urine.

Let us now see what the chemical interpretation of this phenomenon is, so as to be able to explain it. When the deposit has been given plenty of time to form, nothing is easier than to demonstrate its chemical nature: it is composed entirely or almost entirely of earthy phosphates. The phosphates are known to exist in the urine in two forms: phosphoric acid combined with alkalies, sodium and potassium; phosphoric acid combined with lime and magnesium. It is the precipitation of the latter that produces the milky appearance of the urine. The inference might immediately be drawn that patients with milky urine are phosphaturic patients and are to be treated as such; but we shall see that this opinion is erroneous in the majority of cases.

For if we measure the amount of phosphorus eliminated by

these patients in twenty-four hours, the figures will be found normal or below the normal. But may these instances be cases of relative phosphaturia? Let us measure the amount of urea and we shall find that the ratio of phosphoric acid to urea is also either normal or below. In thirteen recent cases of milky urine or phosphatic gravel I found that only four, or 22 per cent., showed absolute or relative phosphaturia; this proportion is small, and phosphaturia seems to me an accessory phenomenon.

The conditions of solubility of the earthy phosphates give us the key of the phenomenon: the earthy phosphates are precipitated because their proportion is too great as compared with the alkaline phosphates, and particularly because the urine is no longer acid enough to keep them in solution. These two causes may be dissociated, or may coincide in the same patient. In the normal state the earthy phosphates represent the third or thirty per cent. of the urinary phosphates, and this proportion may rise to forty, fifty or sixty per cent. Either the acid reaction will be hardly preceptible, or else the urine will be manifestly alkaline, as can be easily shown by litmus paper and at the moment it passes from the bladder. Consequently the two chemical conditions of the phenomenon are an unusual abundance of earthy phosphates, and hypoacidity; phosphaturia is sometimes present also, though it is not at all a necessary thing.

The conditions during which the emission of milky urine is observed are precisely those susceptible of giving rise to these two pathogenic factors. The most important is hyperchlorhydria: excessive secretion of hydrochloric acid by the gastric mucous membrane has as its corollary hyperalkalinity of the blood and, in consequence, of the urine. This modification of the blood serum is generally momentary and limited to the hours that follow the meals, and the milky emissions are also intermittent and appear at the same hours. On the other hand certain dyspeptic patients have a great tendency to excessive disassimilation of their earthy phosphates, and Albert Robin has laid stress on what he calls the earthy phosphaturia of dyspepsia. These two causes can therefore be grouped together.

Next in importance after dyspepsia is neurasthenia, in which the symptoms we are considering are often present. With these cases

the loss in earthy phosphates often predominates, and it is a sign closely connected with their condition. But the milky urine will only appear with them if they belong to the hypoacid group of neurasthenic patients, as opposed to the hyperacid.

With other patients still, hypoacidity is more difficult to explain, but it exists, nevertheless, and gives rise to the phenomenon. A separate category should be made for patients following an alkaline treatment, however slight; with them the explanation is manifest, and their hypoacidity alone is the cause.

These remarks will suffice to show how milky urine may enable us to complete a diagnosis and bring to light the nutritive disorder connected with it. And the importance of the symptoms is equally great through the therapeutic indications that follow it, to which we must still refer.

Milky urine being only a symptom we must naturally treat it by treating its cause, hyperchlorhydria or neurasthenia; but for the very reason that it appears in the course of another morbid state, its treatment must consist in the first place in remedying the earthy disassimilation, and, on the other, the hypoacidity.

We must put a stop to this loss in earthy elements so prejudicial to the system, since lime and magnesium form with phosphorus the framework of our cells. The most suitable remedy for this purpose is arsenic, under any of its forms; while at the same time we must try to furnish the system again, by means of suitable food, with the minerals it has lost. When the loss is particularly marked one of the following wafers can be given at each meal:

℞	Glycerophosphate of lime		
	Glycerophosphate of magnesium, of each	3 grains	0|20

When an analysis of urine, which should always be made in such cases, shows concomitant phosphaturia, the indications are the same, though cod liver oil could then take the place of arsenic.

In the symptom hypoacidity, when it predominates, the best preparation is phosphoric acid in solution:

℞	Phosphoric acid	75 grains	5.
	Distilled water	6 ounces	200.

Mix. S. One or two tablespoonfuls at each meal.

I cannot point to all the particulars of the phosphoric treatment which, like all active treatments, has its precise indications; the reader must turn to the special articles on the subject.

To sum up in a few lines what I have said: Milky urine is met with generally in cases of dyspepsia and neurasthenia and is due to precipitation of the earthy phosphates; the latter condition is caused on the one hand by unusual excess of the bases of the urine, and on the other by hypoacidity; phosphaturia may coexist, but this is not at all obligatory. The two features to be treated are: the loss in earthy bases and the hypoacidity.

LIFE IN THE ANTARCTIC FROM A MEDICAL POINT OF VIEW

BY J. H. HARVEY PIRIE, B.SC., M.B.(EDIN.)

Medical Officer to the Scottish National Antarctic Expedition of 1902–4

As mosquitoes in an uninhabited island could not convey malaria to the voyagers who first land thereon, so the Antarctic regions may be said to be almost incapable of causing any disease in the human frame. Beyond this negative fact, their chief medical interest lies in the effects brought about by the peculiar climatic, dietetic, and social conditions under which life there has to be lived.

I was the medical officer to the Scottish National Antarctic Expedition (1902–4), of which the personnel was 32, all told. The expedition was absent from Scotland 21 months, of which 15 were spent within the southern ice-zone, during eight of these months frozen up in winter-quarters. Of the party all save one returned well, and in better physical condition than on departure.

The health secret lies, of course, partly in the innate salubrity of these regions, but partly also in the careful preliminary preparations which must be taken to guard against the rigors of the climate; and in this connection it may be said the less "sensation" a polar expedition has to record, the better, in all probability, will the outfitting precautions have been.

Personnel.—For such a trip the physical fitness of every one should be above suspicion, and in the selection of the party their social characteristics should also be taken into account. When men have to live in such close companionship for so long, any jarring element in the social atmosphere tells rapidly on the morale of the whole, and hence on the quality of the work performed.

Ship.—The living quarters must be adequately heated. For this a good coal-stove is quite sufficient, and it was found when the ship was well banked up outside with snow that a temperature of 50° F. could easily be maintained inside; but as this meant rather a lack of ventilation, a lower temperature, with fresher air, was preferred.

Sometimes, indeed, it was so low that on waking in the morning it was necessary to break one's self away from the pillow, the moisture of the breath having frozen the beard to it. This condensation of moisture, on the outer walls of the sleeping-births particularly, was a source of considerable annoyance, as during the spells of comparatively warm weather which came from time to time, the ice thawed and made the bed-clothes uncomfortably wet. If there had been no metallic connection with the outside of the ship (bolts, port-hole frames, etc.,) much of this might have been avoided.

Clothing and Sledging.—About the ship we wore simply our ordinary clothes, with the addition of a jersey and rather thicker woolen underclothing than one would wear in winter at home. For general outdoor work, particularly if there was any wind, we slipped over this a wind-suit of a closely-woven gabardine stuff, which is almost impervious to wind and snow-drift. For sledging trips thick woolen homespun smocks, with hoods and knickers and leggings of the same material, were employed. Furs were only used in the form of sleeping-bags. As the lowest temperature experienced when sledging was only —26° F., and as no long journeys were possible, there were no great hardships endured. About 0° F. was found to be the most comfortable camping-out temperature. Above that there was too much melting of the snow below the reindeer sleeping-sacks, and consequent soaking of them from the outside. Below zero, although it began to feel cold, the only thawing to be done was the snow unavoidably carried in about the clothing, and the frozen perspiration and moisture from the breath. All this, of course, soaked into the clothes, which froze hard as boards on emerging in the morning. Such temperatures, however, were never sufficiently low to prevent sleeping. As, after crawling into the sleeping-bag, the flap was always drawn close and toggled down, how breathing was carried on remains to me rather a mystery. One-man sacks were found rather chilly, and the three-man bags were voted warmer than the two-man, particularly by the individual in the middle. Certainly in them there was heat enough to soften foot-gear and gloves put between our bodies; in the single sacks it was necessary to stuff them under one's clothes next the abdomen to enable them to be pulled on again with any comfort.

The hands and feet need particular attention. Two pairs of

Shetland-wool fingerless mittens were generally worn, and on the feet over the ordinary thick woolen socks should be a pair made of goats' hair or of a mixture of goats' hair and human hair, which can absorb a large amount of moisture. Leather boots, although best for ski-ing and generally worn about the ship, become too hard to use on a sledging trip. Either soft reindeer-skin *kumagar* with the hair outside or seal-leather slush-boots are best, and in either case they should be stuffed with the warm, absorbent Norwegian sennal-grass.

Diet.—With the exception of a nearly three months' supply of mutton taken on board at the Falkland Islands and kept in cold storage by lashing up in the rigging, the provisions were largely tinned. Very little salt meat was taken. With the variety of excellent tinned meats now available, and fruits, vegetables, and jams of all sorts, tinned, bottled and dried, a polar expedition can live almost as comfortably as the inhabitants of a land flowing with milk and honey. Samples of the various meatstuffs were examined bacteriologically before our departure by Dr. W. K. Chalmers, medical officer of health for Glasgow, and found sterile. Any tin opened that seemed at all suspicious was at once thrown overboard. Only once was there any trouble caused by the food—two of the ship's company suffering for some days with symptoms of ptomain poisoning from a tin of fish. The plentiful supply of lime-juice on board was scarcely touched, except in the passage through the tropics, but there was never the least trace of scurvy. This may have been due, not only to the careful provisioning, but partly also to the fact that much real fresh food was taken, the resources of the country being drawn upon to the utmost of their limited capacity. Fish were caught in traps lowered through the ice, in sufficient quantity not only to supply the zoölogists with museum specimens, but to feed all hands at breakfast-time two or three times a week, and penguin formed the *pièce de résistance* at dinner five days out of the seven. Although very black and somewhat strong-flavored, stewed penguin was ultimately preferred to any of the tinned provisions, and these birds are so full-blooded that they make delicious Scotch "hare-soup." Seal meat and the flesh of other birds (gulls, petrels, etc.,) were also occasionally eaten. With the return of spring penguin eggs were obtained in thousands and formed a very pleasant addition to the dietary. Throughout the time the appetite of every one was large, but

even in the coldest weather there was never any of that fat-hunger so eloquently described and acutely experienced by Dr. Nansen in his crossing of Greenland. Carbohydrate food (porridge, biscuit, bread and farinaceous puddings) was, however, partaken of largely and served admirably to keep up the body temperature.

Throughout the stay in winter quarters a certain daily minimum of outdoor exercise was insisted on for every one, and the majority went in largely also for ski-running as a sport, in addition to making it the invariable means of getting about. Under this regimen it was found that the body weight, which was taken every month, went up steadily and the strength as tested by the dynamometer showed an increase all 'round.

As we did not winter south of the circle there was even at mid-winter about two hours' daylight and no clinical manifestations were seen of the polar anemia supposed to be due to lack of sunlight and described by, among others, Dr. F. Cook, of the Belgian Antarctic Expedition. From his descriptions I am inclined rather to attribute the symptoms largely to improper nutriment. Hemometric examinations were indeed commenced, but from their negative results and from repeated absences on sledge journeys, etc., they were not kept up.

Surgery.—Two operations were done under chloroform. One was for hemorrhoids, large internal and external piles, which made the man almost useless for work and rendered his life miserable. Cauterizing was the treatment decided on, and if the surgery was somewhat rude it was at least perfectly successful. Through lack of special instruments, a pair of dressing-forceps was used as clamp, and the galley poker as cautery. Healing was quick and quite satisfactory. The other case was an amputation of a terminal phalanx of a finger. Some years before it had been injured in machinery, and it began to pain the man considerably, possibly through implication of a nerve ending in the scar tissue. As it was very misshapen and at best rather useless, it was removed. The wound healed nicely, though rather slowly. It was very noticeable that any cuts or bruises, particularly about the hands, healed very slowly—I suppose because the cold made the blood-supply to the part deficient. But, on the other hand, such a thing as a wound going septic was not seen. This must be attributed to the almost complete absence of pathogenic organisms from these regions.

Bacteriology.—Petri plates exposed to the air for hours and afterward incubated invariably remained sterile. The sea-water, indeed, contained bacteria, but they appeared mainly to belong to the class of denitrifying organisms which play a very important rôle in marine metabolism, but are quite non-pathogenic. That such organisms are not entirely absent was shown by the septic sores and wounds seen on the seals, particularly about the external genitals. Bacterial cultures were obtained from the alimentary tracts of practically every species of seal and bird examined. This is of interest, since Levin has stated that in the Arctic he failed to find bacteria in the intestines of the birds and seals he examined. Although the necessity for the presence of such bacteria in the alimentary tract may not have been fully demonstrated, I doubt very much whether the Arctic animals really have sterile intestines.

On sledging trips we were exposed to cold and damp for weeks at a time and never had a properly dry stitch of clothing on after the first day out, without any ill effects. Surely this is sufficient to prove—if any such proof is now necessary—that cold and damp in themselves are almost harmless. They can merely act as predisposing agents for the invasion of the body by microörganisms. In this connection it may be worthy of note that the ship's party on the return to the Falkland Islands, after an absence of ten months, nearly all suffered from "colds" and gastric disorders, while they had been quite immune all the time "down south." One of the crew had to be operated on at Buenos Aires for appendicitis. Although fully expecting an outbreak of colds, the party left in the Antarctic during the absence of the ship did not suffer at all on the return of the vessel from civilized bacterial regions.

Frost-bite and Snow-blindness.—No serious cases of frost-bite were seen, the worst one, where three fingers were entirely covered by large blisters, like those of a burn, being due to incautious handling of an ice-drill with bare hands. Withdrawal of the fluid and application of lanoline and plenty of cotton wool were all that were required. With the extremities and ears properly covered up, the only parts liable to be affected were the malar prominences and sides of the nose, and as the part becomes quite anesthetic it is always necessary to keep a lookout for white spots on each other's faces. The popular idea of treatment, and, indeed, what is taught in text-books of sur-

gery, is to rub the part briskly with snow. This is about the worst thing that could be done. It may be all right with moist snow, but in temperatures below zero snow is dry and gritty like sand, and rubbing the part with it simply takes off the skin and leaves an open sore. When seen early the application of the warm hand is all that is required, with possibly a little gentle friction; if the condition had lasted long more vigorous massage might be required, and possibly the skin might be rubbed off, but if snow were used it certainly would be.

The worst cases of snow-blindness occurred, not in bright sunshiny weather, but in misty weather, with the sun struggling to break through. Survey work under such conditions proved very trying for the eyes. I had an attack almost limited to the right eye, which I used exclusively peering through a prismatic compass. Tabloids of cocain were employed for the smarting pain, but a day or two in the dark was needed for the condition to pass off.

Medicine.—The medicine chest was not much in requisition. Occasional purgatives and external stimulating applications for rheumatism were the only things for which there was great demand. The rheumatic pains were entirely of the muscular variety and affected some individuals a good deal, but only those who had been subject to them before.

The case of the chief engineer who died of mitral stenosis while in winter quarters, requires some further mention. Although accepted as a first-class life for insurance some little time before sailing, there can be no doubt that he should never have been on the expedition, for his breakdown really commenced on the outward voyage; but he made no complaint for fear, as he said afterward, "he should have been sent home from the Falklands and land us in a hole, short of an engineer." When, from his breathlessness, I discovered his condition it was too late. Although he knocked off work entirely and latterly lived night and day in a deck-chair in front of the cabin stove, the cold proved too much for him. The heart's action was never irregular until near the end, but the contracted condition of the peripheral circulation must have thrown a heavy strain on the cardiac muscle. His strength gradually failed, his digestive powers gave way, then some edema of the legs followed, and there was low delirium for a couple of days before his death. Strophanthus was of practically no avail in improving his condition.

With regard to the use of alcohol, although the crew were signed on "without liquor," it was the custom to serve out a glass of whisky all round on Saturday nights, and also on special anniversaries and holidays. Taken under such conditions—in warm, comfortable quarters—little exception can, I think, be taken to its use, for the little break it made in the monotony of the long winter evenings was very harmless. In the staff's quarters aft alcoholic beverages could be had practically when desired, but it was found as time wore on that the taste for them grew less and less. Coffee and chocolate were much more appreciated as drinks when gathered round the festive board. On sledge journeys one small flask was carried for emergencies, and was once used when, owing to the break-up of the ice, a forced night march had to be made, but it was a whip applied to the jaded horse only when within sight of the winning-post—the ship and warm bunks.

Mentally and socially there was little noteworthy, for one year's isolation from the world is not very trying when all are determined to be cheerful and to take the rough with the smooth as part of the day's work. Work, of which there was always plenty to be found or made, fresh air and exercise, keep the health good mentally as well as physically. Doubtless we suffered latterly a little from what may be termed "mental constipation," owing to a lack of sufficient outside stimulus of fresh ideas. A good general library (which should be a *sine quâ non* on any polar expedition, in addition to the necessary technical works), a supply of games, and some musical talent will prevent any great ennui even in the crew, to whom, of course, the scientific interests appeal less than to the scientific staff.

Two members of the expedition remained on for a second winter under the auspices of the Argentine meteorological service, and kept in very good health, although suffering rather more the second year from digestive disturbances.

Sea-sickness.—A form of sea-sickness which I myself experienced for about ten days, seems worthy of record. After being over a month at sea very severe weather was met with on the voyage northward to Cape Town; as the ship was very light she pitched and rolled exceedingly. Pitching is the worst motion, but rolling is bad enough when it amounts to 56° from the vertical. During that time there was scarcely any of the nausea which usually accompanies

sea-sickness, but there was almost complete inability to retain nourishment. At intervals—the less rough periods—I could get up and enjoy a good meal; for three or four hours all would be well. Then, following on a profuse secretion of thin watery saliva for some seconds, the whole gastric contents were vomited. By lying down I could retain very small quantities of dry biscuit or oat-flour porridge, but almost nothing else. Even in the roughest weather I could, however, take an interest in what was going on or read a book with pleasure. It seemed as if the motility of the stomach was chiefly at fault, but I must confess to not being enthusiastic enough to investigate the character of the gastric contents, as, after vomiting, retching tended to continue unless I laid down at once.

Surgery

THE HYPEREMIA TREATMENT OF SWOLLEN JOINTS

BY E. H. BRADFORD, M.D.

Professor of Orthopedic Surgery in the Harvard Medical School; Visiting Surgeon to the Children's Hospital, Boston, Massachusetts

The surgeon who limits his training to experience in operative cases will find himself at times perplexed when confronted with certain cases of affections of the joints, which, although presenting distressing disability, are not sufficiently active in their symptoms to demand operative interference. When the affections are of a tuberculous nature, or when the bones of the joint are attacked by pus-forming germs, the proper remedial measures have been carefully studied; but in a large class of cases in which the nature of the disease is not as well understood, the question of treatment has been a subject of perplexity to the conscientious surgeon who desires to relieve his patient with the minimum amount of risk and suffering.

A few illustrative cases will serve to define the problems which present themselves to the practitioner.

Case I.—R., an active girl of six years, was brought for treatment with a swollen condition of the right knee. The motion of the knee was unaffected. A slight amount of synovitis was present. But little pain was complained of, although there was some tenderness on deep pressure on the inner side of the joint near the junction of the tibia and femur. On deep palpation a feeling of irregularity could be detected in the walls of the synovial membrane, but no marked loose bodies could be felt, and no bony enlargement. On auscultation of the joint certain abnormal sounds could be detected, but no grating sounds. No enlargement of the bone was present, and no evidence of active disease. After a course of treatment which consisted of compression, fixation, massage, and limited or free motion, alternating according to the activity of the

symptoms, slow improvement took place in the joints, and the condition was such that, on examination five years later, the joint was found to be absolutely normal in appearance, function, and structure. Slight atrophy was present in the thigh just above the knee. The patient, at no time, was obliged to give up activity, or to use crutches. The condition of evident cure has persisted for several years after cessation of treatment.

Case II.—R., a girl of five years, anemic appearance, was presented for treatment, with swelling of the left knee, some limitation of motion, and slight heat. On examination, no increase of synovial fluid was found, but some thickening of the synovial walls. Apparent enlargement of lower portion of the femur and upper portion of the tibia. The patient was brought for treatment before the method of x-ray examination was in use, and, therefore, no skiagraphic record of the condition of the structure of the bone was possible. The limb was fixed with a plaster-of-paris bandage and the patient obliged to use crutches. Six months later a similar condition appeared in the other knee, with some improvement of the symptoms of the knee first attacked. Mechanical support was given to both knees. The patient was prevented from walking actively, but by use of the Thomas knee splints was enabled from the first to move about with some freedom and later was gradually allowed full activity. Treatment by fixation, compression, massage, protection, limited motion, increased motion, and finally free action under careful observation, was persisted in for five years. At the age of fourteen years, three years after the cessation of treatment, the patient was examined and all swelling of the joints had disappeared with the exception of a slight increase in the size of the bone at the lower end of the femur. Function, appearance, and palpitation of the joints were in every respect normal. The patient had grown and was in excellent health.

Case III.—C., a woman, twenty-two years of age, complained of synovial thickening and swelling of the right knee-joint, which had persisted for over a year. She was able to walk, but limped and complained of a feeling of weakness, without pain. She was subject to frequent attacks of increase of synovial fluid and some heat, if the joint was used actively. Fixation treatment and crutches and a plaster bandage was tried for some months with no particular

benefit. The joint was then opened and washed out. Edema, congestion, slight thickening of the synovial walls of the knee-joint, with a very little increase of synovial fluid, but no suppuration, no fringe or loose body or structural changes of cartilage or bone were found. The wound healed readily and the patient again was subjected to fixation treatment, first by plaster bandages, and later a Thomas splint and Thomas caliper splint were tried. A steady but tedious recovery took place, requiring two years before the joint had resumed its normal condition. The patient was finally able to walk without any apparatus or support, and, other than a certain amount of atrophy of the muscles of the lower end of the thigh and corresponding weakness of the ligaments, her condition was normal, and continued so until the last report, two years later.

Case IV.—A., a well-nourished man, of sixty-eight years, has led an active and well-conducted life, with little disability and no illnesses. He has complained of attacks of rheumatism and found increasing difficulty in walking actively, being subject to pain from slight strains. He complained especially of disability in the use of the right knee, brought on, he thought, from slight strain in walking on icy pavements. The condition increased and the patient felt pain on unusual exercise and at times after slight exercise. X-ray examination showed thickening and enlargement in the periarticular tissues, with some alteration of structure of the bones of the joint, indicative of a certain amount of atrophy in portions of the bone and the development of exostoses at the fascial and muscular attachments. Synovial thickening was present and considerable enlargement of the lower end of the femur. The patient improved somewhat after the usual routine office treatment, but finally left home for treatment by baths at a health resort. At the end of four weeks he returned in apparently excellent condition. All pain had disappeared. He had been able upon his return to resume his activity without further trouble, and had continued well on his last report, a year later.

These cases have been selected as illustrative. They are all of the knee-joint, as the knee-joint is particularly typical of the problems which present themselves in treatment. While the etiology of this class of affections is not always known, the pathological anatomy,

both macroscopic and microscopic, has been carefully studied. While there are many varieties and gradations, the changes may be considered, in contradistinction to those joint affections characterized by a destructive osteomyelitis from tubercle bacilli or streptococci or staphylococci, to consist of degenerative changes whereby the tissues of the joints are structurally changed; the elastic tissues necessary to the normal formation of the joint become less elastic, at times more swollen, and later more fibrous; the cartilage becomes absorbed or is transformed into fibrous or osseous tissues, and is followed by the development of irregular fibrous, cartilaginous, or osseous growths, similar changes taking place in the periosteum, in the periarticular tissues, and in portions of the ligaments and tendinous attachments. Atrophy, absorption, and distortion of the bone or abnormal growth follow the impairment of function, the stiffness, and the altered position. (Figs. 1 to 10.)

It is necessary that all scientific treatment should be based upon a sound pathologic basis, and while it cannot be said that the causation is known in all cases of those suffering from swollen joints of this character, yet enough can be learned as to the nature of the affection in most of the cases for a rational deduction as to the best mode of therapeutic attack. In some the process is self-limited to a slight condition of what may be termed degenerative changes, while in others the process continues with a well-defined though slow progress to complete destruction of joint function.

There is a well-marked difference in the gross pathologic changes to be seen when the bone-tissue is primarily invaded as by tuberculous or infectious osteomyelitis from that seen in any of the stages of what may be termed the arthritic group. In the former the epiphysis is attacked by a destructive invading process which is met in the tissues by a separative effort in the attempt to throw off or encapsulate the destructive invasion. In the latter the changes are less acute and seem to be brought about by fibrous changes in the periosteal and periarticular tissues, with fibrous degeneration of the cartilage and resulting changes in the joint tissues, characterized serially by absorption, degenerative atrophy, abnormal and exuberative growth; evidences of subacute inflammation may be present, due more to the irritation of use and friction in joint pressure or joint wear than to an active ulcerative osteomyelitis. The joint

FIG. 1.—Arthritis deformans of the hands and knees, showing atrophy and hypertrophy.

FIG. 2.—Arthritis nodosa of the fingers and hands, showing edema and hypertrophy.

FIG. 7.—Child suffering from polioarthritis deformans, with correction and fixation apparatus.

tissues have apparently gone wrong, and the error of their tissue-building cripples the joint.

An analogy suggests itself in many of these cases with the fibrous degenerative changes seen in the arterial walls suffering from arteriosclerosis or in the condition of the liver in cirrhosis. The condition is one of fibrous degeneration, with resulting injury varying with the function of the organ or tissue.

Clinically the symptoms in the joints are not primarily as acute as are seen when the joint is invaded by pus-forming germs. In a number of cases the subacute symptoms follow an acute painful stage affecting one or more joints, as in articular rheumatism, but in the majority of cases the progress is much more gradual in its development, and the onset is insidious. The motion of the joint becomes slowly impaired, there is swelling of or about the joint, which does not disappear with increasing disability. In the former class well-founded arguments are presented claiming to prove the germ origin of the disease, and in gonorrheal arthritis, and in some other forms, germs are found; in other cases germs of known toxicity are not found and even when germs are present it has been urged that the changes are not the result of direct germ action but of a toxin induced by the presence of the germ, but which may perhaps be also produced by other causes.

In some cases the changes are limited to the synovial membrane, and this may be limited to an edematous or fatty degeneration, and the formation of fringes or loose bodies. The joints are considered to have become dry. In certain stages there may be considerable pain, but, as a rule, the discomfort is that of disability more than of severe pain, but slight pain on unusual motion is common. The patient complains also of stiffness when rising after sitting for any period. Later the affection may slowly develop into the stage of great stiffness and distortion of the limb, with frequent attacks of pain; or the course may be so gradual as to indicate a complete arrest of the process at any stage.

The usual method of treatment of swollen joints has hitherto been to exert pressure upon the swollen parts and to keep the joint at rest. This has been due in part to the general impression that a disabled joint should be kept at rest, that when there is pain there should be a rest of the part, and partly in the belief that when there

is edematous swelling, the tissues should be compressed to counteract the edema of the tissues. The temporary results of this system of treatment are usually good and have warranted its general adoption. But a little reasoning is sufficient to convince the thoughtful surgeon that this method is not only not curative, but may be injurious, if continued for any length of time. Fixation and compression produce a local anemia of the tissues and favor degeneration of tissues. Although the edematous swelling may be reduced, this will return more readily after pressure is removed, and, if the joint is used, the symptoms will be likely to return after a slight strain if the joint has been weakened by the long rest. It is for this reason that the treatment of this class of affections has not been regarded as satisfactory.

Assuming that the swollen condition of the joints is one which is due to sluggish absorption of the tissues about the joint, Professor Bier, of Bonn, has for several years advocated treating this condition by artificially increasing the circulation. It is rational to assume that, if it is possible to stimulate the absorptive power of the circulation around the affected joints, degenerative changes will be delayed or checked, edema will diminish and absorption of the already-developed swollen tissues may be promoted. This reasoning at once raises the question of the possibility of artificially increasing the circulation locally.

This modern method of hyperemic treatment, as it may be called, is not to be confounded with the measures formerly advocated in the days of the metaphysical medicine by the believers in fluxes and peccant humor and the humoral pathology, employing the cautery, the seton, blisters, and counter-irritation, with the idea that the evil should be drawn from the tissues, without which no cure could take place.

Treatment by hyperemia, as it is at present advocated, has a much more rational basis, and, although originally attempted somewhat empirically the initial logical reasoning has been substantiated by clinical results. The treatment can be easily applied. The simplest method of producing local hyperemia is by the application of dry heat, which acts as a local stimulant to the tissues and circulation, dilating the smaller vessels and promoting absorption. "Baking" the limbs, as it has been called, has become generally

Fig. 8.—Radiogram of the hand and wrist of the patient shown in Fig. 7.

Fig. 9.—Radiogram of the elbow of the patient shown in Fig. 7.

FIG. 10.—Radiogram of the knee of the patient shown in Fig. 7.

established as a method of treatment in chronic joints, and various means have been suggested for the application of heat. Of the forms of heat of appliances of many sorts, the simplest is naturally the best, and it is most convenient for the general practitioner to have a cheap and efficient apparatus readily at his command. For this purpose nothing can be superior to the arrangement used by Bier: a light wooden or heavy pasteboard box into which a metal tube is fitted, at the lower portion of which an ordinary Bunsen burner connected with a gas-fixture can be placed. A lamp of kerosene or alcohol can be used in the place of the gas-burner, although the heat cannot be as readily controlled by the latter as by the former. More elaborate forms, with electricity as the means of furnishing heat, are found in use in establishments where an extensive outlay is possible, but for the ready use of the practitioner not much elaboration is necessary. It is manifest that if the enclosing box is of a material which limits radiation less heat is needed, and a more diffused heat can be employed. The heat should be completely under the surgeon's control. It should surround as far as is possible, be equally distributed around the limb or the part affected, and not be intense at one limited portion.

It has been suggested that there may be some virtue in the rays from an electric light, which may have a therapeutic effect not furnished by the ordinary dry heat. This possibility, however, cannot be regarded as more than a supposition, as there are not sufficient facts to justify such an opinion. It should be regarded as probable that heat is the chief therapeutic agent.

The amount of heat which is to be applied varies with the individual and with the conditions. It is, of course, unnecessary to apply enough heat to desiccate the tissues, but it is desirable to furnish enough heat to produce an active perspiration. Attempts have been made to surround the limb by an absorptive material which would withdraw the perspiration as soon as it is formed (by moisture-absorbing gas or cotton) and thereby to increase the evaporation and stimulate the local circulation. This, however, may be regarded as probably an unnecessary elaboration, it being merely essential that the air-space surrounding the limb, or part to which it is applied, be sufficiently large and sufficiently hot to allow an ordinary amount of evaporation. The amount of time that should be given to

the baking of the limb is a matter of judgment, and cannot be prescribed generally. The limb should be baked at least for twenty minutes at a sitting, and the sitting should be either daily, twice a day, or more frequent, according to the severity of the symptoms. It is a practice of some surgeons to protect the limb by wrapping it

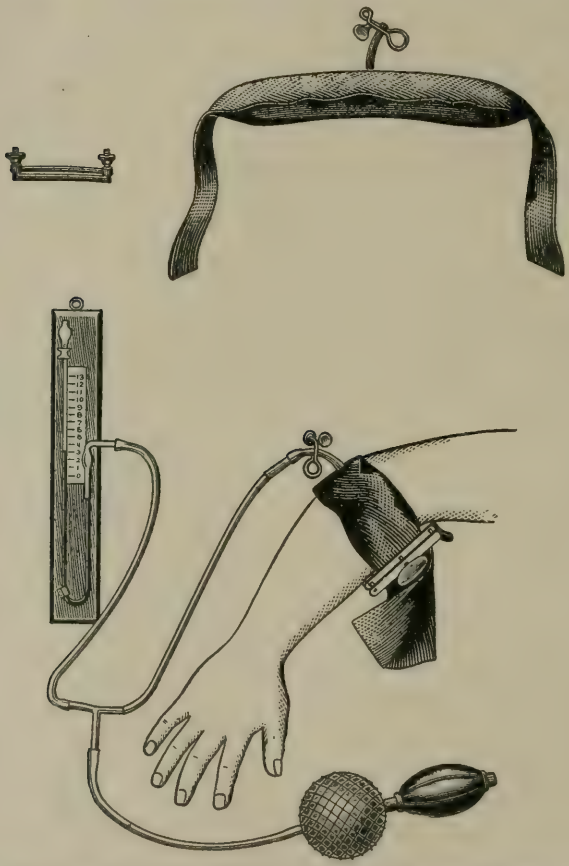

FIG. 13.—Apparatus for producing constriction hyperemia, with index of pressure; also inflatable air-cushion, constricting band, and clamp.

in a woolen cloth, thus giving an opportunity for the entrance of greater heat into the cabinet. It is probably preferable, however, that an arrangement be made by which the heat is not applied with that intensity as to cause discomfort, and held in an enclosure where, radiation being checked, a gradually increasing heat may be given

FIG. 11.—Apparatus for securing hyperemia of the shoulder by means of dry heat.

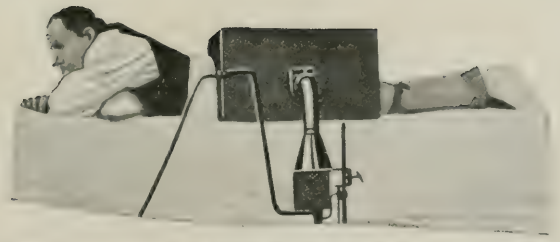

FIG. 12.—Apparatus for securing hyperemia of the trunk by means of dry heat.

the joint. To use kitchen terms, the joint is to be subjected to a "slow" rather than a "quick" fire. The accompanying illustrations (Figs. 11 and 12) will be sufficient to indicate the simple forms of portable heat appliances. When a number of joints are involved the patient can be placed in a cabinet or in an arrangement enclosing the greater portion of the body, and allowed to remain for

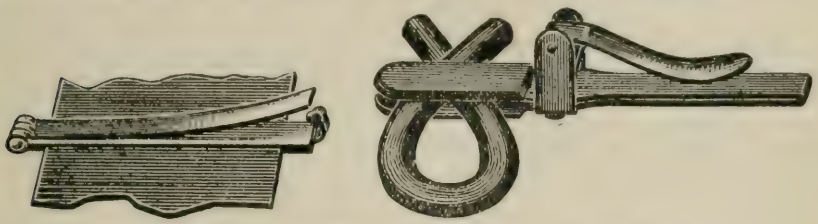

FIG. 14.—Clamps for constricting elastic bands and rubber tube.

some time in this position. When a large portion of the body, however, is treated in this way it is necessary that the patient should be carefully watched, the pulse carefully observed.

Another form of developing hyperemia of the tissues artificially can be furnished in the following manner: An ordinary cotton bandage is applied from the foot to just below the knee, if the knee

FIG. 15.—Stasis (passive) hyperemia for the arm and hand.

is the joint involved, and an elastic band is wound above the knee sufficiently tight to prevent the venous flow of blood toward the trunk, but not sufficiently so to prevent all arterial circulation; a condition of passive congestion will follow. This, of course, can be carried to any extent, but it is unnecessary to apply this further than to produce a slight change in color of the skin, and it should not

be applied to such a degree as will cause a diminution of the normal local temperature. It can be applied without a bandage below the part affected, simply using a constricting elastic band above. This same method can be applied to all of the joints, but while it is difficult to apply constriction to the hip or shoulder in such a way

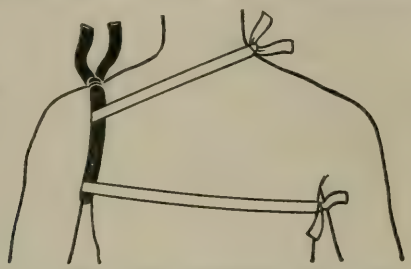

FIG. 16.—Stasis (passive) hyperemia at the shoulder.

as to produce as complete anemia as at the knee, enough can be done at the former joints to produce a considerable congestive hyperemia. It is better ordinarily to apply this form of hyperemia mildly for several hours, rather than for a short time with greater compression.

FIG. 17.—Stasis (passive) hyperemia at the hip.

It will be found that on the removal of a constrictive elastic band an increased flow of blood follows, and it is possible to furnish what may be considered circulatory gymnastics with the purpose of subjecting the tissues to an active stimulation by the repeated applica-

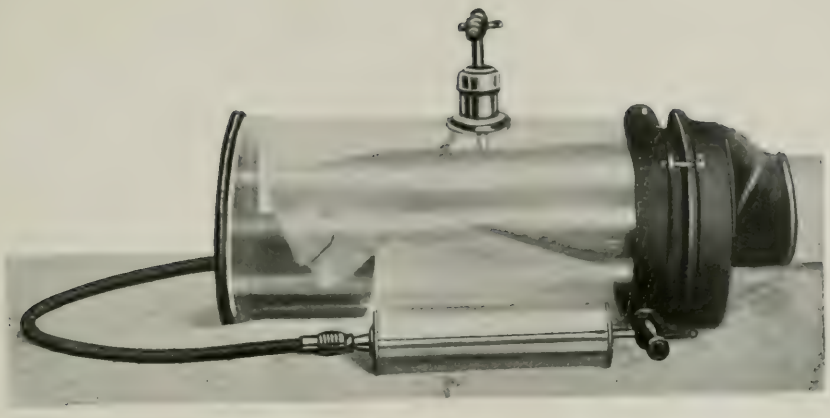

Fig. 18.—Apparatus for securing vacuum hyperemia.

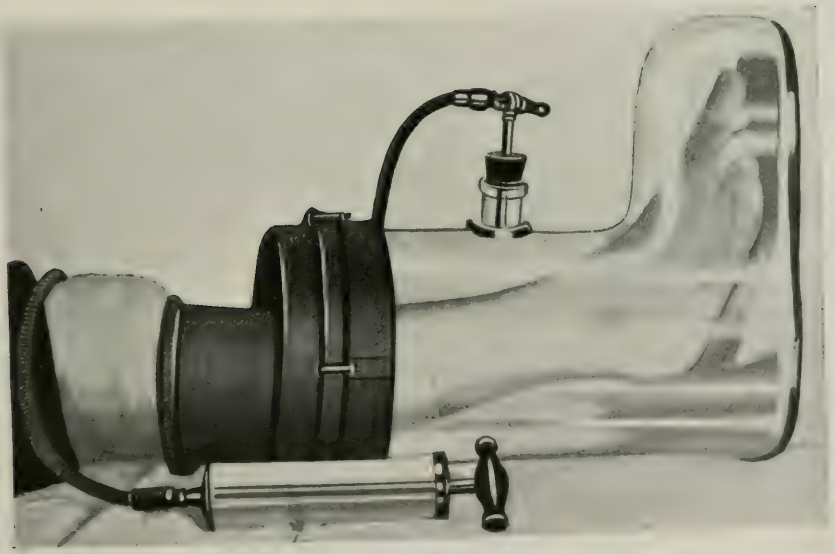

Fig. 19.—Apparatus for securing vacuum hyperemia of the foot.

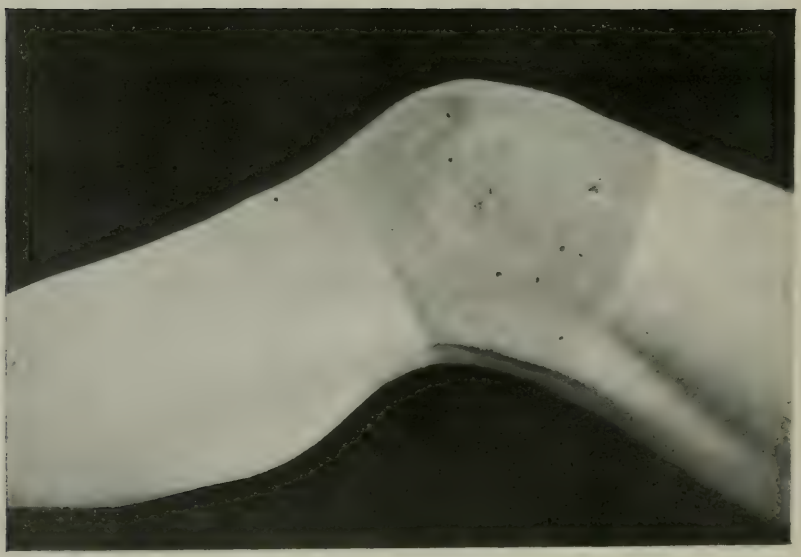

FIG. 20.—Capillary extravasations due to the excessive use of passive congestion.

tion and removal of a tightly-applied compressive elastic bandage. It is evident that this requires considerable judgment, as there is a certain amount of danger of capillary extravasation, if congestive hyperemia be carried too far. (Figs. 13 to 17.)

Still another method may be employed for artificial hyperemia that may be found of advantage, namely, that of placing the joint or the limb in an enclosed metal or glass chamber, where the air may be attenuated. The illustrations (Figs. 18, 19, and 20) indicate the way in which this can be applied, and the method will be found of use. It is difficult to employ this without a certain amount of constriction, for the reason that it is impossible to produce a vacuum without some compression around the limb, which necessarily produces a degree of congestive hyperemia. This hyperemia is increased by the suction produced by the vacuum, and the method may be considered as one of active hyperemia combined with a limited amount of stasis.

An alternation of anemia from increased atmospheric pressure, with hyperemia from suction, would furnish the most thorough possible circulatory gymnastics, but the mechanism required is too elaborate for general use.

The use of vacuum on inflamed tissues is not a new one—it is perhaps almost as old as medicine—but Bier's arguments are new, claiming that there may well be a reaction in practice against the hitherto well-accepted belief that all inflammation must be combatted by the abstraction of blood from tissues; that the proper antiphlogistic measures could not be heat or congestion. In surgery the poultice and hot cloths have been used from time immemorial, and in ununited fractures several surgeons, notably Thomas, of Liverpool, have sought to create an increased amount of blood in the part to be treated; hydrotherapy and the douche have accomplished this, but to Bier belongs the credit of having formulated the previous ideas and presented the theory in a practical form, to the effect that under certain conditions, treatment should consist in the employment of the readiest means to congest the tissues.

It is of importance to determine exactly the effect upon the tissues produced by heat, by stasis, and by vacuum hyperemia, and to learn whether these measures simply act upon the circulation of the blood, increasing directly or indirectly the amount or flow of

blood in the affected parts, or whether any influence is brought to bear upon the lymph or the cellular activity or cell conditions.

Can the living tissues be chemically changed or "cooked" by the prolonged application of endurable heat? Are the phagocytes stimulated? Is immunity in this way favored protecting against the influence of toxin? Is the transmigration of white corpuscles increased, or are the changes simply those resulting from the greater activity of the arterial circulation? All these questions, although of interest and importance, can only be partially answered, as our exact physiologic knowledge on the subject is not so extensive as is desired, but enough facts are known to justify rational conjecture.

To determine as far as possible the effect upon the tissues of this application, experiments have been made by Drs. Low and Z. B. Adams, at the Boston Children's Hospital laboratory, and through their courtesy I am allowed to quote the following facts from a preliminary report of the investigations, which are still in progress: The shaved leg of a guinea-pig was subjected to a hot-air bath for fifteen minutes; the guinea-pig was killed, the tissues were placed in a fixative, and cross sections made which were subjected to microscopic examination. Other limbs were subjected to constriction and to suction, and the tissue sections were examined in a similar way. These sections showed a dilatation of the capillaries and small vessels in the tissues treated by heat; the arteries were more enlarged than the veins; where stasis and suction hyperemia had been applied, the same enlargement of the capillaries was seen. In stasis hyperemia the veins showed more enlargement, and there appeared to be an increased migration of the white corpuscles more than where suction or heat alone had been applied to the limb. Further investigation will probably show that if the treatment is prolonged there is in suction hyperemia also a dilatation of the lymph-channels, that in heat hyperemia there is an increase of local heat, and it is reasonable to suppose an increase of cell change, that the tissues are stimulated to a renewed activity.

These experimental facts confirm the evidence obtained in clinical experience, which shows that hyperemia treatment is useful in relieving the symptoms in certain cases, when an improvement in the circulation is to be desired.

The frequency with which treatment by hyperemia should be applied, the length of time for each sitting, the selection of the form of congestion to be employed, are questions which can only be answered in individual cases. When the method of dry heat is used, it is advisable that the application of the heat should be begun at a comparatively slight elevation of temperature, with a gradual increase, until the amount of heat applied is as much as can be borne by the patient. It is important that the heat should surround the joints affected equally, and should not be directed especially to one joint. It is for this reason that the confined space surrounding the limb should be neither too large nor too small—large enough to allow a certain amount of diffusion of heat, not so large as to permit a waste of heat, nor so small as to give too intense a heat. When a number of joints are affected and a considerable portion of the body is exposed to heat at the same time, the patient should be watched carefully, and the pulse constantly recorded. When congestive hyperemia is to be applied care must be exercised, as it is possible to apply the remedy in such a way as to cause capillary hemorrhages, especially in old people. The rule should be that the application should be made to such a degree as will not cause a blue color of the skin or coldness of the surface. Suction hyperemia should also be applied with moderation, and cannot be continued for the same length of time as congestive hyperemia.

Other tried measures for stimulating the circulation, and thereby promoting absorption, are not to be overlooked in this condition. Both massage and hydrotherapeutics have long been proved to be of value in a chronic faulty condition of the tissues. So much has been written on these subjects and they are so well known that nothing more than a mention is necessary here, except to add that they serve as excellent adjuvants to the measures described, each having its proper applicability. Massage will always be a valuable means of improving the local circulation, as it is readily applied and it requires no greater skill than is generally at command. Hydrotherapeusis also furnishes an excellent stimulant to the general circulation, but hot dry air, favoring evaporation more readily than wet applications, is more efficient in causing local hyperemia. With a combination of ice massage (*i. e.*, rubbing the part affected with a piece of ice) and the hot-air bath, the surgeon is able to increase to

almost any desired degree the amount of blood at any portion of the body. A useful aid will be found in the application of the well-known electric vibrator, which is an arterial stimulant of considerable value. Although the exact physiologic effect of the vibrator is not known, it is evident that it may be made to have an evident effect upon the vasomotor system. The same may be said of electricity, especially of static and high-frequency electricity.

It is evident that in these cases it is not sufficient to be satisfied with the alleviation of the local symptoms of swelling. In many cases after the primary cause of the affection has been removed, disability remains even after the swelling has diminished. A faulty condition of the muscles and articular tissues from simple disuse interferes with the activity of the limb. Movement at and the use of the joints should be allowed, except in the active stage of tuberculous epiphyseal ostitis, or in the acutest stages of all joint affections. Rest is indicated in the acute stages, but the stage of fixation is of much shorter duration than was formerly believed. Protected and graded movement may be not only harmless but beneficial in the later stages of joint affections.

When the joint movement is checked by the changing of a few periarticular ligaments motion can be gained by forcible straightening, but ordinarily the process of recovery of motion is necessarily a more gradual one; the tissues which have undergone fibrous degeneration require time for the transformation of non-elastic to elastic tissues. For this purpose graduated exercises are arranged, beginning with easy movements, with a gradual increase of the arc of motion in such a manner that the violence of the motion does not excite muscular resistance. This is the principle of the so-called movement cure, which can be arranged by complicated or by simple apparatus. Daily exercises can be given to the patient which can be performed by himself or by the aid of a well-trained, experienced nurse, competent to move the joints without too great violence or exciting muscular opposition. In certain instances the function of the joints is interfered with by fringes or loose joint bodies, and the condition can be improved by their removal. But in degenerative processes the trauma incident to operation is to be considered and the tissue disturbance incident to dissection undertaken less readily than when a joint is suffering from destructive ostitis. No

rule can be laid down as to the proper limits of rest and motion, or as to the use and abuse of retaining and supporting splints, as these questions are matters of judgment in each case.

It is not expected that this or any method will be of permanent benefit in the advanced cases of what may be termed "ruined joints." No miracle can be performed by the application of local hyperemia, in cases in which advanced destruction of tissue has already taken place. Distortions and anchylosis demand the appropriate operative treatment, a description of which does not come in the scope of this paper. But even in advanced cases, symptoms due to local congestions can be relieved by the employment of means which relieve congestion, and it has been abundantly proved that permanent cures may be effected in well-developed cases by the persistent use of proper remedies. The following cases may serve to illustrate the fact that certain of the cases may make a satisfactory recovery under conservative treatment:

Case V.—W., a rapidly-growing boy, sixteen years of age, presented two chronically swollen knees, with a slight increase of synovial fluid. At times the joints were larger than at others; occasionally there was slight pain and some heat on palpation. Irregular projections and fringes could be felt within the cavity of the joint. There was some laxity of the joint tissues. The boy had grown rapidly and was not strong, but was otherwise well. The motion at the knees was normal, but the boy was limited in his activity on account of a sense of weakness at the knee. Treatment was carried on with slow improvement, but recurrent attacks occurred with increasing infrequency for a long period, requiring treatment during and following the attack. Eventually the knees made so complete a recovery that at the age of twenty-one he was able to enter upon a career demanding strength and activity. Treatment was discontinued and renewed according to the symptoms, and later the case was under observation for a number of years. The treatment consisted of compression for a short time, massage and graduated exercises. When last seen slight laxity of periarticular tissues could be felt and some irregularities suggesting fringes around the patella. The joints were free from any disability whatever, but the left knee was slightly larger than normal.

Case VI.—H., a child of four years, was seized with a painful

swelling of the left knee, causing distortion. Same condition was observed in ankle of same limb, and later the knee and ankle of the opposite side, together with the joints of the elbow, the hand, and the fingers, even the temporo-maxillary articulation, were attacked. The joints were in such a painful condition that massage was impossible, and could only be given in limited form. Few joints escaped, and the child remained for two years in a pitiful condition of helplessness. The only treatment that could at first be applied was that of correction of deformities and fixation of the limb. As the child was placed upon a frame change of air was possible. Child's condition gradually improved, and six years later one knee-joint had recovered nearly normal motion. Stiffness, however, remained in the joints of left hip and right knee, also anchylosis in right elbow. In many of the joints complete recovery took place, the motion of the jaw, left elbow, left knee, became normal; the motion of the fingers was free, but the wrist on the right side was somewhat stiffened. At the age of twelve the child became able to walk and attend school, all active symptoms having disappeared, though disability remained in the above-mentioned joints.

Case VII.—P., a girl of nineteen years, was taken with a swelling of the knee without pain. Periarticular tissues were affected with slight synovial affection. This condition remained for four years, gradually improving, with treatment of fixation and protected use. A swollen condition of the left knee developed to a slight degree. The condition gradually improved without treatment, and five years later patient had become nearly well, had married, and later gave birth successively to three healthy children. Before the birth of the second child a slight swelling of the right knee-joint was noticed, persisting for several months. It subsided, and since the patient's limbs have remained in excellent condition.

Case VIII.—B., a younger sister of P., sixteen years of age, was taken with swelling of left knee, followed by swelling of ankle, subsequently by swelling of right ankle and right knee, with swelling later of both elbows and of left wrist, with some irritation of the arteries of the dorsal region. A gradual painless stiffening and disability of the joints followed, with gradual increase for over a year. Under treatment of congestion hyperemia and application of dry heat, followed by the use of graduated exercises, there was a

complete recovery of the motion and appearance of the right knee and of the left knee with the exception of a slight enlargement. Some limitation of motion of ankle remained, and the elbows gained in motion, but with the persistence of some limitation of motion. The wrist-joints and some finger-joints remained distorted. The patient is still under treatment for the condition of the hands and right elbow, but with increased improvement.

From clinical experience it is evident that in the treatment of swollen joints the surgeon has a number of remedial measures besides operative or constitutional treatment, which are applicable for the purpose of relieving such local conditions of the joints as are unfavorable to recovery. Among these remedial agents the surgeon can rely on the hyperemia method as certainly of no less value than the means hitherto more generally used, fixation, pressure, etc.

THE SURGICAL COMPLICATIONS OF PNEUMONIA AND THEIR TREATMENT

WITH A REPORT OF FOUR CASES OF EMPYEMA: ONE DISCOVERED IN A CADAVER, ONE POINTING IN THE GROIN, AND SIMULATING A PSOAS ABSCESS; ONE IN A CHILD TWO MONTHS OLD; AND AN INTERLOBAR EMPYEMA *

BY T. TURNER THOMAS, M.D.

Assistant Surgeon to the University and the Philadelphia General Hospitals; Assistant Instructor of Surgery in the University of Pennsylvania

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Like the disease itself, the surgical complications of pneumonia are due to infection, and usually result in pus formation. Thus we may have an empyema, an abscess or gangrene of the lung, or an abscess in some other part of the body, as a metastatic extension from the pus collection in or about the lung. For example, Claytor [1] reported a case of multiple brain abscess following empyema, and collected from the literature 58 cases of brain abscess resulting from primary lung disease. Brain abscess from pulmonary disease is not very common, and is more likely to result from lung disease than disease of the pleura, although it may be due to either. Empyema as a cause, however, was second only to bronchiectasis.

Abscess of the lung is most commonly due to pneumonia, and when due to this cause gives the most favorable prognosis. Murphy says that many of the cases reported in the literature as lung abscesses are actually the result of empyemas and subdiaphragmatic abscesses, communicating with a bronchus. Abscess of the lung is often difficult of diagnosis, the condition sometimes being first suspected on the sudden expectoration of large quantities of pus, which will be the more significant if fetid. The pneumonia does not subside with the usual crisis, and a septic condition sets in with chills, sweats, hectic flush, etc. Localization may be difficult, especially when the abscess is small and deeply situated. If there is no communication with a bronchus the dulness may be localized by per-

* Read before the North West Medical Society of Philadelphia, March 5, 1906.

[1] Philadelphia Medical Journal, March 2, 1901.

cussion, and in the presence of such a communication signs of a cavity may be obtained. Exploratory puncture for diagnosis cannot be employed with the same safety as in empyema, because of the danger of escape of pus and blood into the free pleural cavity when adhesions are absent. The presence of adhesions is exceedingly difficult to determine by an external examination. The skiagraph is sometimes of value in localizing the collection.

The treatment consists in exposing freely the surface of the suspected portion of the lung, by resecting one, two, or more ribs. Exploratory puncture may then be employed for localization. Digital palpation is said to be very valuable for the same purpose. When the abscess has been localized the pus may be evacuated with a Paquelin cautery, a blunt pair of forceps, or a knife. In the presence of circumscribing adhesions the abscess may be evacuated, drained and dressed immediately. When they are absent, the free pleural cavity must be protected from infection either by suturing the visceral to the parietal pleura around the opening in the chest wall, or by shutting off with gauze. Frequently the surgeon proceeds at once to open the abscess, but it is preferable to wait twenty-four or forty-eight hours for the development of adhesions, provided the urgency of the case does not demand immediate evacuation.

The most frequent surgical complication of pneumonia is empyema, and as in abscess of the lung, of all the causes of empyema, pneumonia is the most favorable. Like all serous membranes, the pleura is very sensitive to irritations and responds quickly and actively to infections. As in similar involvement of the synovial membranes and peritoneum, the first effect may be a pouring out of its serous secretion, which may later be absorbed or become purulent. It is probable, however, that in most cases the fluid is purulent from the start. As in all so-called serous cavities, normally there is no actual cavity, the corresponding serous surfaces being apposed to each other. Each lung fills its allotted space in the thorax, and through its elasticity and the incoming and outgoing air, the lung accommodates itself to the varying capacity of the chest in respiration. When, however, an excess of pleuritic fluid accumulates, it does so at the expense of the normally apposed structures and in the direction of least resistance. The rigid chest-wall remains firm, while the soft elastic lung yields, like a sponge, to the pressure of

the collecting fluid, and according to the quantity of the effusion is more or less functionally incapacitated. The increasing pressure later displaces the more resisting structures, the diaphragm and abdominal organs are pushed downward, the heart to the opposite side, the intercostal spaces bulge, and the whole chest-wall on that side becomes fixed in the position of inspiration. Usually the diagnosis of an empyema may be made readily upon the recognition of these mechanical results, the dulness and absence of normal lung sounds, the evidence of sepsis, and in the cases under discussion the history of the preceding pneumonia. The condition, however, is often overlooked, because the attending physician fails to examine his patient for the physical signs, and does not note the change in symptoms from those of a frank pneumonia to those of sepsis. There may be very little on inspection to excite the suspicion of the examiner.

While working as a prosector to Dr. G. G. Davis, of the University of Pennsylvania, I was forcibly impressed with this fact during the preparation of an anatomical specimen, by the formalin injection method, to show the positions of the thoracic and abdominal organs in situ. The specimen, after about four years, is well preserved and is among Dr. Davis's collection of anatomical specimens at the University (Figs. 1 and 2). The body of a colored man was selected, because of its apparent normal size and shape. It was well developed, without excess of fat, and almost perfectly symmetrical. Upon removing the anterior wall of the thorax and abdomen a very large empyema was discovered on the right side. It would appear that the man died with the condition unrecognized, as nothing, evidently, had been done to relieve it. To a wide-awake physician there must have been, during life, evidence enough of its existence.

When an empyema is recognized early, and is evacuated and drained, the lung, although collapsed at first, soon tends to expand, and usually within a short time is again in contact with the chest-wall. When, however, the condition has existed long enough, the lung in its compressed position develops a thickened inelastic pleura and strong adhesions, so that it fails to expand after the fluid has been evacuated. Much retraction of the affected side and a permanent cavity and fistula result. In order to obliterate this cavity extensive multilating operations, like the Estlander and Schede,

FIG. 1.—View of the cadaver, showing the line of incision in the anterior thoracic and abdominal wall.

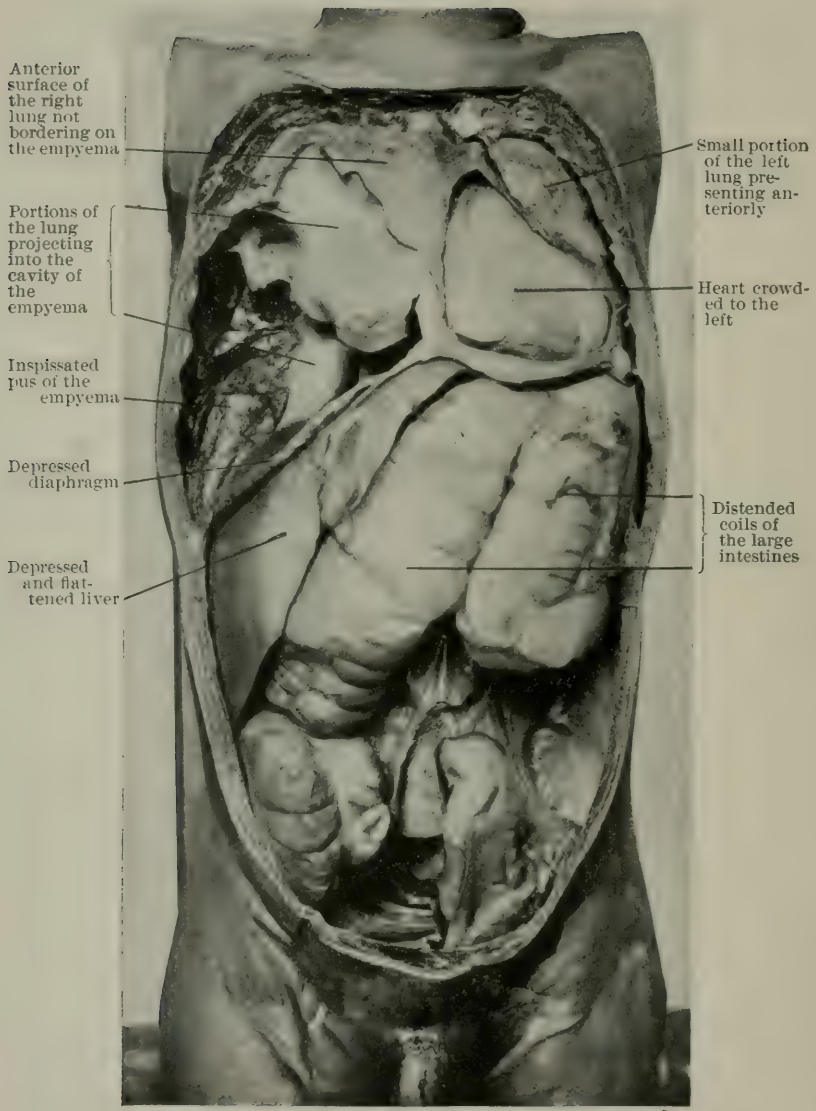
Anterior surface of the right lung not bordering on the empyema
Portions of the lung projecting into the cavity of the empyema
Inspissated pus of the empyema
Depressed diaphragm
Depressed and flattened liver
Small portion of the left lung presenting anteriorly
Heart crowded to the left
Distended coils of the large intestines

FIG. 2.—showing the position of the thoracic and abdominal organs as influenced by the empyema.

with or without removal of the visceral pleura as recommended by Delorme, must be done. Early diagnosis and operation are, therefore, of great importance to promote the rapid and full expansion of the lung and healing of the wound.

Undoubtedly some cases of pneumococcic empyema get well without any operation, and others after simple aspiration without drainage. Without operation, however, the absorption of the exudate, if it occurs at all, is very slow and incomplete, the pleura becomes thickened and indurated and in some cases calcified. The tendency to contraction and deformity is thus increased, while the process may light up again at any time. In case absorption does not occur and operation is not done, the pus sooner or later escapes spontaneously through a bronchus, through the chest wall (empyema necessitas), or it may burrow downward through the diaphragm to become a perinephric abscess, and as in a case operated on recently by Dr. J. William White, in the University Hospital, it may point in the groin. Osler [2] says "very remarkable cases are those which pass down the spine and along the psoas into the iliac fossa, and simulate a psoas or lumbar abscess." The history of this case is as follows:

The patient was a laborer, 26 years old, and was admitted for a fluctuating swelling in the inguinal region, which was at once diagnosed as an abscess. He says that he was never sick until two years ago, when he had an attack of "pleurisy" lasting about three months. He lost weight and strength, had night-sweats and coughed considerably. After the attack had lasted eight weeks, his physician tapped his chest below the scapula on the right side. Only about an ounce of pus was evacuated. He expectorated considerable thick yellowish sputum for about three weeks after the chest was tapped, and in a few months seemed to have regained his former health. Ever since this sickness, he says, he has had "rheumatism" in the right hip. He had to stop work twice for about three weeks at a time. When asked to describe the seat of this hip pain, it is found to be rather in the region of the sacroiliac joint and is somewhat vague.

About two months before admission he first noticed a small lump in the groin about the size of a walnut, and he says that there has

[2] Practice of Medicine.

been very little pain and tenderness about it. It has steadily increased in size until now it is about the size of a closed fist. He is a fireman and says that the lump bothers him at his work, more from its size than from any pain or tenderness associated with it. The exploring needle showed yellowish creamy pus. The urine was normal, likewise the temperature, pulse, and respiration. At the operation about a pint of creamy yellowish pus was evacuated. The abscess cavity extended upward between the ilacus muscle and the peritoneum beyond the reach of the finger. The cavity was rapidly closing when he left the hospital.

Cantley,[3] after an analysis of 86 cases of empyema in children treated by various methods, makes an interesting comparison of the results. In four cases no surgical treatment was employed. One died, and in the other three there was much subsequent contraction,—a mortality of 25 per cent. In 12 cases aspiration was the only treatment. None died, but there was no recorded case of complete and perfect recovery, although such may have occurred and not have been recorded. There were, however, 40 of the whole series, in which aspiration was employed, 28 requiring further surgical treatment. Incision and drainage without resection was the treatment adopted in 35 cases, of which 18 were aspirated first; 28 recovered and seven died, a mortality of 20 per cent. Of 35 cases in which resection of a portion of a rib and drainage was the treatment used, six died, a mortality of 18.2 per cent. While the results of simple incision and resection were practically the same on the whole, a comparison of the results at different ages showed that the mortality among patients treated by resection gradually rose with the diminution of the age of the patient. The mortality from simple incision remained the same whatever the limit of age taken. Of five cases under two years treated by resection, four died, a mortality of 80 per cent., while of four cases under two years treated by simple incision, one died, a mortality of 25 per cent. The comparison of the relative advantages of incision and resection, however, lost much of its significance from the fact that many of the patients died from causes unconnected with the operation.

It is generally recognized that the resiliency of the bone in the

[3] Internat. Med. Mag., June, 1895.

very young permits a sufficient separation of the adjoining ribs to allow the introduction of a good-sized drainage tube, and, therefore, free drainage. Judging from Cantley's observations, and so far as my knowledge goes, from the literature generally, in very young infants simple incision without resection is sufficient. The only case in which I have employed this method was that of an infant two months old, who died probably as the indirect result of the method of operation. That empyema at such an age is very rare, may be inferred from the fact that of 400 cases of empyema in children, reported by different authors, there were only 10 under one year, and only one under two months of age. Cantley reported 86 cases under 12 years, T. Wardrop Griffith 28 under 12 years, Momson 48 under 10 years, and Caille 80 under five years. Weightman reported 124, and Batten 48 cases, in which the age limit was not given, although the fact that they were children indicates that they were not much older than 12 years. Of the 10 under one year in only four was the age in months given. Momson lost one at eight months, while Caille gives the age of his three as one month, five months, and six months respectively, but gives no further details of the cases. Since the histories in these young infants are so scarce and my case presents some interesting points, it will be presented here with some detail.

In June, 1902, I was asked by the late Dr. W. O. Xander to operate on this patient at its home. There had been some difficulty in having her admitted to a hospital because of her age. The pneumonia developed at six weeks and the empyema was recognized at two months of age. Aspiration was tried first and six ounces of pus were removed. The improvement which followed was marked but temporary, and four days later an incision was made in the sixth interspace about in the middle of the axilla. A considerable quantity of pus escaped. Without much difficulty a rubber tube with a lumen three-eighths inch in diameter was introduced to just within the pleural cavity and sutured to the wound edges by silkworm-gut. An abundant sterile dressing was applied and changed daily without irrigation.

During the following three weeks the child improved considerably. The cyanosis about the eyelids, lips, fingers and toes, which had been present before the operation, disappeared, and the tempera-

ture fell from 104° and 105° F. to about 100° F. Owing to the weakness of the pulse and the uncontrollable movements of the patient the pulse was never satisfactorily counted.

For the next few weeks no improvement in the temperature or general condition was noted, and although the appetite was apparently good, the mother strong and healthy, and her supply of breast milk abundant, the patient not only did not gain in weight but was gradually losing it. The discharge from the wound was slight. Six weeks after the operation the drainage tube was removed and a gauze drain substituted. The temperature which, while the tube was in, remained persistently at 100° F., now began to subside, in a few days was normal, and the child was more at ease. As the tube had reached just within the ribs, an attempt was made to close the wound. The gauze was introduced to a slightly less depth each day, and in a week was withdrawn altogether. A few days afterward the temperature rose to 100° F., the child cried a great deal, and the breast was refused. After the removal of the rubber tube the ribs, which had been kept apart by the tube, gradually approximated each other, so that with the granulation tissue they made the introduction of the gauze by the probe somewhat difficult. When the wound had been permitted to close and the patient grew worse, it became necessary to reopen the drainage track with the probe and to reintroduce the gauze, which consisted of a very narrow strip. It was here that the chief difficulty arose. Owing to the narrowness of the space and the obstruction to the probe and gauze, any sense of resistance at the bottom of the drainage track could not be appreciated. It was, therefore, practically impossible to determine how far the probe should be passed, and later to ascertain the degree of healing that had taken place within. The post-mortem later showed that the probe and gauze, evidently, were responsible for the failure of the wound to heal. The necessity for a larger opening was clear, but the parents could not be induced to permit a resection of a rib.

On the day following the reopening of the wound and the reintroduction of drainage, the temperature fell to normal. Soon afterwards it was noted that the cough had ceased entirely, the tongue was moist and clean, showing that the patient, evidently, was not septic and was not suffering from other lung disease. The loss in weight persisted, however, in spite of forced feeding by various milk

mixtures in addition to the breast feeding, all of which the child seemed to tolerate very well. Two prominent pediatrists examined the child at this time, but could not determine the cause of the failure in nutrition. So far as I could see, there was nothing to prevent the child getting well, yet it persistently failed in nutrition until it died 15 weeks after the operation.

Permission was obtained to open the chest on the affected side. The lung was found in contact with the chest-wall and diaphragm everywhere and adherent except in the region of the apex. Nothing abnormal could be detected on palpation of the lung nor on inspection. The sinus encroached on lung tissue and was about two inches long from the internal surface of the ribs. There was no cavity associated, but immediately about it a small area grayish white in appearance.

The case illustrates a difficulty associated with simple incision through an interspace and drainage in these young infants, which I have not seen mentioned elsewhere. While the ribs could easily be forced apart sufficiently to permit the introduction of a good-sized drainage-tube, the pressure on the tissues induced by the tube seemed to have been sufficient to keep up the temperature, since it almost immediately fell to normal when the tube was removed. Again, the same resiliency which permits the ribs to be forced apart, readily, and to be kept apart by the tube, allows them to come together soon after the tube is removed, and thus leave a very poor opening for drainage or for judging the conditions inside the chest. In view of the fact that the patient was not septic and evidently was not tuberculous, another cause must be sought for the persistent emaciation. That the patient was capable of a healthy reaction was shown by the prompt general improvement after the first removal of pus by aspiration, by the second improvement after incision and drainage, by the fall of the temperature to normal after the removal of the tube; and when the temperature had risen on the closure of the wound, by its prompt return to normal after the drainage-track had been reopened and redrained, with the presence in a short time of a clean, moist tongue, good appetite, and the cessation of the cough. All this, in my opinion, argues that a larger opening was indicated and therefore a resection of a portion of a rib, which would have been less dangerous than an insufficient drainage

opening. Another point suggested to me by this case concerned the situation of the opening. In such an infant the sitting position will rarely be assumed. After the operation because of the necessity of renewed activity of the affected lung it will be best to keep it as much as possible on its back, whether it is in the mother's lap or in bed. If a resection were made as far posterior as possible, just external to the thick longitudinal muscles of the back, there would be provided almost continuous free and dependent drainage, and a wound that would require little interference. I believe that the small opening in this case necessitated a degree of irritation which the immature tissues and delicate nervous system could not tolerate.

Since the days of Celsus resection of a rib has been done for the treatment of empyema, and while other methods have been employed, with more or less success, it is the treatment of choice with the majority of surgeons to-day. Free drainage is the rule in all pus collections, and while the opening provided by the usual resection is very small, it is much freer than can be obtained by an incision through an interspace. If the empyema is localized or encysted the position of the drainage opening will vary according to the situation of the collection, as determined by the exploring needle. When it is not localized it is best to get as dependent drainage as possible. Schede and C. Israel resect the tenth rib, thus bringing the opening to about the lower border of the lung, which corresponds to the tenth thoracic spine. In children it is sufficient to resect the ninth rib.

The incision may be curved with the formation of a flap, or, as I prefer, the simple straight incision over the rib may be employed. The periosteum, with the surrounding structures, is separated from the rib by a periosteal elevator, thus avoiding hemorrhage from the intercostal artery, and an inch or an inch and a half of the rib excised by a bone-forceps or a Hey's saw. The pleura is then opened, the pus evacuated and the index-finger introduced for exploration and for the removal of masses of lymph. Irrigation will rarely be necessary. Two large rubber tubes should be introduced, one often becoming plugged by fibrin in the interval between the dressings, and should be sutured to the edges of the wound to prevent their falling into the thoracic cavity. It is well to pass a safety-pin

through each tube across the wound, for fear the sutures should cut through the tissues. Both tubes should be removed from time to time for cleansing, and if at any time the patient shows signs of increasing sepsis the tubes should be examined to see if they are blocked.

Sometimes the pus collection is situated in one of the fissures, and if deeply placed may be difficult to localize. Such an interlobar empyema was reported by Dr. J. H. McKee and me before the Pediatric Society last May, in which the presence and location of the pus were nicely demonstrated by Dr. McKee. The opening in the chest-wall, made by a resection of about one inch of the sixth rib in the midaxilla on the left side, led directly into the cavity. That the empyema was a localized or encysted one was shown by the fact that the lung was adherent to the parietal pleura in the circumference of the empyema, which was demonstrated by the exploring finger. That the pus collection was in the fissure was evident from the depth of the cavity, which was approximately in the normal situation of the fissure dividing the two lobes of the left lung; and from the fact that the finger came directly upon the heart. The bottom of the fissure, normally, is in relation with the lower portion of the heart. Drainage was provided by two good-sized rubber tubes, introduced in the direction of the spine for about five inches. These were gradually shortened and healing was completed in five weeks. At the time of the operation the finger entered a roomy cavity, the walls of which were, apparently, under the influence of external atmospheric pressure. Soon afterward it could be determined through the wound by the eye and the finger that the cavity was becoming filled by expansion of the collapsed portion of the lung, this being aided by coughing and forced respiratory exercises. In a few weeks the cavity was completely obliterated, and soon afterward the wound closed.

A PRELIMINARY NOTE ON A RATIONAL OPERATION FOR THE RADICAL CURE OF INGUINAL HERNIA

BY A. N. M'GREGOR, M.D., F.F.P.S.G.

Assistant Surgeon to the Royal Infirmary, Glasgow, Scotland

The operations for the radical cure of hernia are many and varied, complex and simple, similar and diverse; like the members of a botanical group, they have a family resemblance, with enough difference to warrant individuality, and the application of a name. Some of these operations are really beautiful in conception, some are daring interferences with Nature's plan, and others are ingenious methods of overcoming certain difficulties, but all have for their aim and object the cure of the defect—the hernia.

The very existence of so many methods of treating hernia is a proof of the difficulty of obtaining routine success. It indicates that the true, complete, and perfect operation is not yet; and the constant influx of new operations suggests a search for the unattainable—an operation which will be suitable and successful under all the varying conditions of size, site, and anatomic relations that are peculiar to hernias. It is hardly possible that one method of treatment can cover every possible variety of inguinal hernia, but it is none the less true that a proper appreciation of the elements that make for success is necessary, and that our knowledge of these is, as yet, incomplete. With the view of aiding and forwarding research in this direction the following considerations are submitted, together with their application in an operation which is suitable for the majority of cases.

Stated briefly, the objects of any operation for the radical cure of hernia are: (1) The return of the protruded viscus to its proper domain within the abdomen, and (2) the adoption of such measures as appear suitable and sufficient to keep the abdominal contents within the abdominal boundaries. In order to attain this second requirement it is recognized that due attention must be given, (*a*) to the disposal of the hernial sac, and (*b*) to the obliteration

of the passage through which the hernia took place. The elements of success are variously accredited, by their authors, to be modes of treatment of the sac and to the ingenious dispositions of the fasciæ, or combinations of both. It is principally in the latter,—in what might, indeed, be called the engineering difficulty,—that most ingenuity is seen, that the greatest diversity of opinion exists, and for which new solutions are suggested.

If the general question were raised as to the cause of failure, the natural reply would be that the hernial openings occur at the lower part of the abdominal wall which may support some of the weight of the abdominal contents, that a natural weakness exists there, that in inguinal hernia the presence of the spermatic cord is a serious obstacle, etc. These and many other reasons are commonly advanced, and each has its value, but there is one which has hitherto been neglected, which forms a necessary part of many procedures, and is therefore worthy the attention of surgeons—the separation of the areolar tissue which lies in intimate contact with the fasciæ.

Contrast a Bassini or a Halsted operation with the "through-and-through" suture of the abdominal wall as successfully practised by many gynecologists and others, and bear in mind that these procedures have for their object the closure of an opening in the abdominal wall and that through similar tissues. The question arises, Why should an opening in one part of the abdomen require all this rearrangement of fascial layers in order to prevent the recurrence of a hernia, when other openings in the immediate vicinity, and through similar structures, are closed without hesitation and by simple means?

The answer to that question, for the most part, is an anatomical one. The muscles of the anterior abdominal wall are continued to their insertions as tendons and sheets of fasciæ. These sheets of fibrous tissue contain no blood-vessels except those derived from the overlying and underlying connective tissue. Schäfer [1] may be taken as an authority. He states that the blood-supply of tendons and fasciæ is derived from the surrounding and penetrating layers of areolar tissue, "the fibrous tissue receives blood-vessels, but they are inconsiderable both in number and size as compared with the mass

[1] Quain's Anat., Ninth Ed., vol. 2, pp. 56 et seq.,

of tissue to which they belong. The lymphatics, however, are in great abundance in the enveloping areolar sheaths of tendons and aponeuroses. In addition to these, a close network of lymphatic vessels may be injected in the deeper parts of the tendons where they run in the alveolar septa."

It will be conceded, then, that the tendons and fasciæ are dependent on their sheaths or covering layers for their nourishment; it follows that the removal of the over- and underlying areolar tissue from the fibrous insertions of the external oblique, internal oblique, and transversalis muscles is the most efficient way of depriving these structures of their blood-supply, of their lymphatic connections, and of their nerve-supply. It is no exaggeration to say that this reflection of the areolar tissue and the separation of the layers of fasciæ form part of almost every operation for the radical cure of hernia, and I venture to state that, proportionately to the extent of that separation, the healing of the fascial wound is delayed. It is evident that the restoration of the blood-supply is a necessary preliminary to satisfactory union of the layers of tendon and fascia, and when it is remembered that the union of areolar tissue with the fibrous tissue must be cicatricial, and that the newly-formed blood- and lymph-vessels must pass through that cicatricial tissue, it seems obvious that the vitality of the tendons and fascia must be considerably impaired. So, too, the severance of nerve-fibers must be detrimental. This, then, is the main cause of failure and of relapse in the radical treatment of hernia—excluding of course sepsis, faults of technic, and gross congenital imperfections of the anterior abdominal wall. The success of the many methods of fascial manipulation is the main argument against this contention, but I submit that the success of these methods is due to the forcible detention of the displaced fasciæ, by sutures of considerable resisting power capable of maintaining the parts in position for a long time,—*i.e.*, some time longer than four weeks, until sufficient adhesion takes place; that the effects are produced and maintained by these mechanical means rather than by the early union of the tissues; and that, as these structures are comparatively inert, vital union does not take place for a considerable time. It will be evident, too that if, during this long period of delayed repair, any accidental loosening, separation, or tearing of the sutures takes

place, relapse is inevitable; and that if, instead of the suture itself tearing, the fascia be split by the tension of the suture and the superadded strain of vomiting or coughing, the result will be unfavorable.

The preservation of the areolar tissue seems to me to be the point that counts most in estimating the causes of successful repair of laparotomies as against the results in hernia. The incision for an intra-abdominal operation goes straight through the abdominal wall, the layers of which are not separated, and so, when the edges of the wound are properly brought together, repair takes place readily, for the blood-supply of the tissues to be united has not been impaired. In hernial operations the surgeon usually cuts down to the fascia and then strips off the areolar tissue and satisfies himself by ocular demonstration of the pillars of the ring, intercolumnar fascia, Poupart's ligament, etc. It seems to be a habit of the dissecting-room days, a clearing away of the fat, as a preparation for the more intricate part of the operation, and though it is necessary in many of the modern operations, it seems to me an unnecessary and detrimental measure in the majority of cases.

The element of time is an important one. It may be assumed that the healing of the skin wound is no criterion of the state of repair of the underlying tissues after a Bassini, Halsted, or other flap-splitting operation; and it has already been said that the maintenance of the fasciæ in place for something over four weeks is due to the sutures and not to the vital union of the tissues. To quote a recent text-book of operative surgery:[2] "Where it is possible to carry out the following rules, they will be found exceedingly satisfactory: For a period of four weeks after operation, rest in bed. For a period of six weeks after operation, when the bowels are being moved the patient should be in the recumbent position so as to avoid strain on the wound. For a period of three months after operation there should be no hard manual work."

In the operation to be described, which is suitable for the great majority of cases, the patient may be allowed to sit up in a fortnight and may be dismissed in three weeks.

[2] Binnie, Second Ed., pp. 421 et seq.

Operation.—An incision is made over the inguinal canal, from a point corresponding to the middle of Poupart's ligament, forward and inward to the pubic spine. It divides skin and areolar tissue and, at its inner end, exposes the external abdominal ring and the structures passing through it. The sac is separated from the elements of the cord and returned by the Macewen method. The treatment of the sac recently described by Nicoll, or any other method which results in the formation of a plug, buttress, or bouchon, at the internal abdominal ring, should suffice. The skin and the areolar tissue is left as little disturbed as possible. In order to facilitate the suturing and, in some cases, for other reasons such as the adhesion of the neck of the sac, it is advisable to cut open the whole length of the canal. The cord is gently stretched by traction on the testicle and at the same time it is separated from its normal position and pushed toward the upper margin of the canal. The sutures are introduced at intervals of about half an inch, the first being placed close to the inner ring. The sutures are inserted from above downward and the needle, in succession, passes through areolar tissue (subcutaneous), fascia of the external oblique or edge of conjoined tendon, emerges in the canal in front of the cord, passes in and out of the posterior wall of the canal to emerge below it, just above Poupart's ligament, then through areolar tissue. The other two or three sutures are inserted similarly, the last one closing the pillars of the external abdominal ring. These sutures are tied with moderate firmness and result in the slack of the canal being gathered into folds which lie lengthwise of the canal. The suture is outside of the cord, which occupies a new position closely covered with fascia—a new canal. The loose fasciæ and connective tissue are so folded together that in 14 days' time they feel like a hard cord which lies parallel to the spermatic cord. When it is possible to keep the anterior wall of the canal intact the sutures are guided by the point of the index-finger in the canal; it is preferable to the open method, though more difficult of application. As the needle passes into the canal from above the finger should cover its point and then push the elements of the cord upward behind the needle, the point of which now engages the posterior and inferior walls and its point appears under the skin of the lower lip of the wound.

For all cases in which the canal or a portion of the canal is recognizable the above operation can be readily and quickly performed with great security. When the external and internal rings are widely distended and opposite each other, and in cases in which the abdominal wall is defective in its fascial development or by trauma, special procedures are necessary; these will be discussed in a more complete report when the results have been collected and tabulated.

Stated briefly, the objects of this operation are to shorten the time of operation and so minimize shock and the immediate after-effects; to lessen the period of the patient's after-treatment; and to restore him to work in a shorter time than is safe with most other operations.

In order to attain these objects the areolar tissue is retained in contact with the fascia whose blood-supply, lymphatic connections, and nerve-supply, it carries, so as to favor the reparative process and to simplify the operative procedures. Further, the lumen of the canal is diminished so as to accommodate only the spermatic cord; the excess of fascia is gathered into a cord which lies parallel to the spermatic cord and therefore end-on to the course of an advancing hernia, from which it is further protected by the pad of gathered-up sac over the abdominal aspect of the inner ring.

The results have been very satisfactory in those patients who have been seen at varying intervals after operation, though it is not wise to generalize on this point until a more complete search has been made for those patients who have not reported themselves.

INGUINAL HERNIA IN THE FEMALE

BY E. SCOTT CARMICHAEL, M.B., F.R.C.S.(EDIN.)

Assistant Surgeon to the Royal Hospital for Sick Children; Gynecologist to the Royal Public Dispensary, Edinburgh, Scotland

ALTHOUGH inguinal hernia is more common in the male sex, and the femoral variety in the female, one is likely to overlook the fact that the former is of very common occurrence also in the weaker sex. Statistics show that in adults this form of hernia occurs in the female in from 5 to 10 per cent. of all cases. In 184 radical cures for this variety of hernia in children that I have done, 23 were in females; while of a total of 729 radical operations in both sexes treated in the hospital, 76 were in little girls,—giving a percentage of 10. I am indebted to Mr. Stiles for permission to make use of his cases in addition to my own.

The relative frequency of this hernia does not seem to vary with age in the two sexes to any appreciable extent. For the sake of comparison, however, in children under puberty, the cases occurred at the following ages:

21 were under 1 year of age,
21 were under 5 years of age,
34 were over 5 years of age.

The ages here represented denote the time at which the radical cure was performed, and although the history of the date of the appearance of the hernias is unreliable, it would seem probable that the majority of cases were noticed within the first few years of life and were probably of congenital origin. Inguinal hernias in male infants and boys are in the majority of cases congenital, and one is justified in concluding that this form of hernia in female children is also congenital.

As in the male, inguinal hernia is more common on the right side, for of the above cases 39 occurred on the right side, 24 on the left side, and 13 were double.

Contents of the Sac.—Contents were found lying in the sac or were pulled down into the sac in 30 cases, the relative proportion be-

ing as follows: Fallopian tube and ovary, 17; Fallopian tube alone, 7; small intestine, 2; omentum, 2; cecum and ovary and Fallopian tube 1; and vermiform appendix and ovary and Fallopian tube, 1.

In two cases the sac was tuberculous, the clinical examination revealing in each case a nodule, resembling in size and shape the form and character of the ovary.

Hernia of the Ovary and Fallopian Tube.—It is suggestive that the persistence of the so-called canal of Nuck should be associated in such a large proportion of cases, over 30 per cent., with malposition of the ovary and Fallopian tubes.

An examination of the normal position of these organs in the pelvis of the young child reveals a most striking variation. In many instances the ovary lies longitudinally alongside the lumbar vertebræ, on the psoas muscle almost as high up as the lower angle of the kidney, while in others it lies on the false pelvis, in the cavity of the true pelvis, and in some cases just inside the internal abdominal ring.

In how far the descent of the ovary is dependent on the action of the gubernaculum or inguinal ligament is difficult to estimate, but that this band is in some way responsible for the position of the ovary is probable. According to McMurrich, the descent of the ovary is due to the slower growth of the inguinal ligament as compared with the abdominal wall, which must lead one to suppose that ovarian hernia is due to a retarded development of this band.

Of 28 cases in which any content was present in the sac, the ovary and Fallopian tube are present alone or along with another viscus in 24 cases. That the frequency of their presence can be explained by simple mechanical causes is extremely unlikely, and one must rather consider some association with the development of the inguinal ligament as the more probable explanation.

In the truly acquired inguinal hernias of the adult female the presence of the ovary is then probably a mechanical one.

Various authorities, such as Englisch, Bitzako, and others, give a higher percentage of acquired as compared with the congenital form of inguinal ovarian hernia, but its frequency in early childhood and its close relationship to the canal of Nuck would lead one to conclude, as in the male sex, the probable congenital origin of many that might otherwise be considered acquired.

Whether the formation of this canal is to be considered analogous to the processus vaginales of the male, is doubtful. According to Duplay, Beumier, and others, this canal does not exist as a normal protrusion, and although Martin, Waldeyer, and others, consider it to occur normally as in the male sex, the consensus of opinion would seem to support the former.

One is inclined after operation in the above series of cases to consider that the so-called canal of Nuck is an abnormal protrusion, associated in some way with the development of the inguinal ligament, and caused by the same conditions as lead to a drawing down of the ovary and Fallopian tube. The ovary and Fallopian tube do not lie free in the sac in the same sense as a loop of intestine or omentum, but are intimately associated with its wall, the broad ligament or mesosalpinx springing directly from the posterior wall. The length of the canal seems to have a certain bearing on the presence or absence of the ovary, as it is rarely present when this is short, but is nearly always present when the canal is long and extends well down toward the labium majus. This probably is due to an abnormal shortening of the inguinal ligament.

Lockwood seems to imply that ovarian hernia results from abnormal lengthening of the mesovarium and mesosalpinx, and although in a case recorded by him in which there was torsion of the ovary this may have been present, one is unable to support this view from the present series of cases. The attachment of the mesentery to the posterior wall of the sack was frequently short and often markedly sessile, so as to render replacement of the organs of considerable difficulty without first dividing the peritoneal folds. It seems more probable that the frequent presence of the ovary in inguinal hernia is due to the drawing down or protrusion of that portion of the peritoneal covering of the pelvis which gives attachment to the broad ligament, and is in some way due to the inguinal ligament.

The relation of the round ligament or lower part of the inguinal ligament to the canal of Nuck is an interesting one. Waldeyer believes that the peritoneum covers the round ligament above and in front, but does not surround it, while Devreux considers it to be attached to the apex of the so-called canal of Nuck.

In the cases operated upon the round ligament appeared distinct up to the apex of the sac, and then seemed to spread out and become lost on its exterior. Puech believes that these hernias are due to the traction so exerted by a shortened round ligament.

A large percentage of inguinal hernias in the female are bilateral, and this tendency seems to be marked the younger the subject. In babies under one year, of the 21 cases 8 were double; in babies under 5 years, of the 21 cases 2 were double; in babies over 5 years, of the 34 cases 3 were double.

In adults, on the other hand, the bilateral condition would appear not to be so frequent. In some cases the hernia of the opposite side is not noted till after radical cure has been performed on the one side.

Another point of interest in connection with these hernias, is that the younger the subject, the more often is the ovary and Fallopian tube likely to be present in the sac. Again referring to the ages: In 21 under 1 year, ovary and Fallopian tube in 13 cases; in 21 under 5 years, ovary and Fallopian tube in 7 cases; in 34 over 5 years, ovary and Fallopian tube in 2 cases.

It is only fair to state that the ovary and tube were not actually present in the sac at the time of the radical cure, but in some cases by pulling on a fold of peritoneum, namely the base of infundibulo-pelvic ligament, constantly present in the sac, the Fallopian tube followed by the ovary came into view.

Lockwood in a case recorded refers to the relative position of the ovary and Fallopian tube, in which he describes the former as lying lower than and in front of the tube. Operation on the present series of cases does not bear out this view, as the tube almost invariably appears first in the sac.

Again, while it is rare to find the ovary alone in the sac without the Fallopian tube, in 3 of the above cases the tube lay alone in the sac, considerable traction being required to expose the ovary.

In order to replace the ovary and Fallopian tube within the abdomen, it is necessary to divide freely its attachment to the sac, and that brings one to consider whether it is advisable to remove the ovary, to prevent future trouble, and to what extent herniated ovaries differ from the normal. In order to satisfy myself on this point, I have examined a large number of children post mortem to ascertain

the normal condition of the ovary, not only as regards its position, but with reference to its general microscopic appearances.

As above referred to, the position of the ovary varies very markedly in different subjects, and presents in addition, as regards its size and shape, the most striking differences in subjects of the same age, and at different ages.

It may be long, flat, and almost ribbon-like in one instance, while in a child of the same age it may be thick and short, resembling a small bean, while it may vary as to its surface from being smooth and homogeneous to one presenting considerable irregularity from the presence of small follicular cysts. With such evident variation in the normal condition, one is chary in removing any herniated ovary in children. I have not yet felt justified in doing so, and cannot give any evidence as to their microscopic appearance.

Birnbaum in a recent paper describes the histologic appearances of the ovary removed from the hernial sac of an adult. The ovarian stroma would appear to be less dense than normal and to contain collections of cells taking the form of ducts and bands, and in some cases resembling follicles. These follicles are lined by cylindrical epithelium, the interior often being filled with the same kind of cells. Well-formed primordial follicles were not present.

In other cases recorded, the ovaries presented the appearance of acute or chronic ovaritis, cystic degeneration and new growth formation.

Although these changes may be present, many cases are recorded in adults, in which the herniated ovaries functionated, swelling at the menstrual periods, and even uterine and ectopic pregnancy have frequently been observed in these cases.

With such clinical evidence of the persistence of ovarian function, one is not justified in removing such ovaries, at any rate as a routine practice.

In the congenital forms of hernia, there are frequently associated malformations of the other genital organs. Sterility in the adult is generally present in double ovarian hernias, while absence of the uterus, uterus bicornis, bipartitus, pseudo-hermaphrodism and true hermaphrodism according to Olshausen are not infrequently present.

Hernia of the Uterus.—The uterus itself may be herniated into the canal of Nuck. I have not observed this in any of the above

cases, although in one instance on traction of the ovary and Fallopian tube, the uterine horn appeared at the internal abdominal ring. Hernia of the uterus has been recorded not infrequently. Recent papers by Jophson [1] and Helgerrenier [2] state that it has been recorded in over forty cases, in eight of which the uterus was pregnant. It appears to occur most frequently in the adult, in which it seems to be associated generally with multiple pregnancies, or with hermaphrodism.

The uterus itself presents usually some malformation, such as bicornuate condition. This is most frequent in the congenital forms as seen in children. It is probable that more than half the cases are really congenital, and that in the majority some defect or malformation of the genito-urinary tract is present.[3]

It seems to occur most frequently on the left side, as opposed to the frequency of inguinal hernia in general, which is most common on the right side, and is generally associated with hernia of one or both tubes and ovaries, and small intestine.

Hernia of Bowel and Omentum.—From the frequency with which inguinal hernia occurs on the right side, the cecum and appendix is a not infrequent content. Although it is probably the most constant portion of bowel found in right-sided hernias in the males and more especially in young children [4] the large sacs seen in these hernias in the male are rarely seen in the female. In the two cases of the present series the cecum and appendix were only partially present.

The part of the small intestine, when present, is usually as in the male, the lower part of the ileum just above the cecum. This

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[1] Annals of Surgery, 1904.

[2] Berliner klinische Wochenschrift, March 1906.

[3] Küstner and others have recorded most of the cases known.

The non-pregnant uterus is generally found in a hernia of congenital origin, and impregnation may occur in a uterus so placed, or the uterus when pregnant may pass down into a preëxisting hernia When it occurs in the adult, multiple pregnancies are a predisposing factor, especially if a hernial sac is already present, and sudden emotion, as in two cases reported by Lallement in washer-women, is an important exciting cause. Rouffárt, Leqmen and others record cases of hernia of the uterus associated with imperforate vagina, a condition apparently common in one congenital variety. Pseudohermaphrodism is recorded by Brohl.

[4] British Med. Journ., Feb. 3, 1906.

was present in the sac in two cases. The omentum also was present alone in two cases.

Tuberculosis of the Sac.—Tubercle of the sac of a hernia is not infrequent, occurring in about 3 per cent. of hernias in young subjects. It may be entirely unsuspected or may be diagnosed before operation. It generally occurs in one of two forms: First as a tuberculous nodule generally toward the apex of the sac. In this form it resembles not infrequently, in size and shape, the ovary, and except for its somewhat less marked mobility is difficult to distinguish. It is an isolated nodule of many, lining the parietal peritoneum of the abdomen, and is, as a rule, associated with tuberculous glandular enlargement of the mesenteric glands. It is well in all cases in which such an oval swelling is felt in a hernia to examine carefully the abdominal cavity for evidences of glandular enlargement. The most commonly affected glands are those situated in the lower end of the enteric mesentery and ileocecal junction. This form of tubercle of the sac is the most favorable although even in such cases a guarded prognosis must be given.

In the second variety there is a diffuse infiltration of the sac, which is thickened and converted into tuberculous granulation tissue. No part of the sac is healthy. Examination of the abdomen reveals no abnormality, and there is often little if any glandular enlargement. A recent case operated upon in private practice was that of a child aged eighteen months, which had been in good health until shortly before operation. It had been noticed to be out of sorts, not taking its food so well for a week or two before I saw it, but not sufficiently so to induce the parents to call in a doctor.

The child was well nourished. No thorough examination was made of the abdomen before operation. Radical cure was performed of a right-sided hernia. The sac was markedly tuberculous, presenting the appearances of the second variety. The abdomen was then examined while the child was still under the anesthetic, but no glandular enlargement, ascites, or distension was present. A guarded prognosis was given to the parents. The child recovered from the operation, but six weeks later developed tuberculous meningitis with a fatal termination.

The peritoneum of the abdomen in these cases appears wholly

converted into a lining of tuberculous granulation. There may be no adhesions and no clinical evidence of its presence until discovered at the time of operation.

Of the two varieties of tuberculosis of the sac, the first is the more common, and can, as a rule, be diagnosed before operation.

Clinical Features and Differential Diagnosis.—When a swelling is present in the inguinal region in a female subject, there are several possibilities that occur to one in making a diagnosis. When a hernial sac contains bowel, as a rule the diagnosis is easy. The characteristic symptoms of a hernia of the intestine are present.

A condition which is not infrequently met with, is the history of slight swelling in one inguinal region. On palpation an oval swelling the size of a bean is felt. Pressure in the adult will usually elicit the peculiar sickening feeling if it is the ovary, but in the child no such sensation, if present, helps the diagnosis.

Such a swelling may be presented by a variety of conditions: (1) A superficial lymphatic gland, (2) the ovary in the sac, (3) omentum in the sac, (4) an encysted hydrocele of the canal of Nuck, and (5) a tuberculous nodule in the sac.

The ovary, when present, is usually mobile, and can be displaced up into the canal, while a gland has not, as a rule, so much mobility in the direction of the inguinal canal.

An encysted hydrocele of the canal of Nuck is usually only slightly mobile and is not a common condition.

A tuberculous nodule simulates very frequently the ovary. It may be distinctly mobile and is generally situated at the apex of the sac. It is always well to examine the general abdominal condition in all such cases, although a diffuse peritoneal tuberculosis may be present, without affording any evidence on examination of the abdomen.

Hernia of the omentum is, as a rule, irreducible and may in some cases resemble in form that of the ovary. It is not common in children, occurring much less frequently than in the adult female.

I have not intended in the present paper to discuss the treatment. The only satisfactory method of cure in almost all cases is by operation, which may be undertaken at any age from birth upward. The earlier the sac is removed, the less will be the weakness in the inguinal region in adult life, a point of considerable importance to the child-bearing woman.

THE CLINICAL SIGNIFICANCE OF PERITONEAL ADHESIONS

BY CHARLES GREENE CUMSTON, M.D.

Surgeon to the Floating Hospital, Boston

The pathologic anatomy of the formation of adhesions has been elucidated principally by Graser. He demonstrated the formation of peritoneal adhesions in animals by placing two peritoneal surfaces tightly in apposition by means of layers of lead suture. The microscopic examinations of preparations obtained in this way gave excellent pictures when stained with the Weigert fibrin stain. This showed that the peritoneal endothelium, before reaching the stage of firm adhesions, undergoes retrogressive changes, and, by the progression of these changes, a thread-like development of fibrin occurs on and about these cells, which originally covered both the peritoneal layers, and signs of degeneration become more and more marked.

A secretion of this type always occurs between the edges of the wound, or between the adherent serous surfaces, although only to a slight degree in many cases. At these points the degenerating endothelium supplies the ferment in order to give rise to fibrin formation from the fibrin generators of the inflamed plasma. They consequently represent centers of coagulation. At the end of the second day vascularization of the fibrin meshes occurs, and, at the same time, along the course of the neighboring capillaries spindle-cells arranged in the form of a bow develop, and it is from these that the new-formed vessels develop. From his most recent work, Graser concludes that adhesion and cicatrization are only possible after the death of the endothelium of both layers. There must be some preliminary process in Graser's experiment, which involves the endothelium in the first place, while the two serous surfaces are in contact, before adhesions are possible.

Heinz, who comes to the same result in his experiments, says that the endothelium of the serous membrane protects the peritoneum

from the formation of adhesions, and that these were only developed when the endothelium had been removed from some cause. We, therefore, may conclude that the formation of adhesions is briefly as follows: Necrosis and desquamation of the endothelium of two approximated layers of serous membrane; from this an agglutination occurs in consequence of an exudation of fibrin; and finally a definite adhesion is developed, because the fibrin becomes organized. The intensity of the fibrin formation, which always exists between two adherent serous surfaces, and the extent of the development of new-formed vessels, are, as has been demonstrated by Graser in a very large number of experiments, in proportion to the intensity of inflammatory reaction of the parts irritated, when this irritation is due to infection or chemical agents.

In adhesions, which are produced by strictly aseptic means, the fibrin exudate, and later the appearance of new-formed vessels, and so forth, is not very marked. On the other hand, this will be considerably greater in adhesions which result from the pressure of two serous surfaces one upon the other when robbed of their endothelium, upon an intact serous surface, or one which has been infected or roughly handled.

In direct connection with this is to be considered the toughness of the adhesion. The above-mentioned form of adhesion is capable of little resistance and can be easily broken down, while the latter type, on the other hand, possesses a considerable amount of resistance, because, after the occurrence of organization in the newly-formed substance, there is always a tendency for the latter to contract.

Clinical observation reveals the same condition of affairs, and every surgeon is familiar with the slight bands of adhesion which are interlaced together and can be easily broken down with the finger without difficulty, while in other cases, as, for instance, when an intense inflammatory process has arisen in consequence of pus formation, the hardest, toughest, and most callous adhesions will be found, which can only be separated by means of the scissors.

The time necessary for the formation of adhesions is often extremely short, as many clinical experiments show. Fritsch is of the opinion that a few minutes are quite sufficient. But at all events, the possibility of operations upon the gastro-intestinal tract rests

entirely upon the possibility of very rapid adhesion formation in order that union between the openings in the gut or stomach may take place; for, if the ability of rapid and sure adhesions between the serous surfaces, did not exist, that old standby, the seroserous suture of Lembert, would have remained an impossibility. The coalescence of the adhering surfaces naturally is a longer process, but this nevertheless takes place, as has been proved by the experiments of Graser and Heinz, within forty-eight hours. Macroscopically, the adhesions soon form delicate meshes which spread out between the various viscera of the abdominal cavity, and then transform into tough bands which finally become flattened and firm, varying in this respect according to the intensity, the seat, and the extent of the process which was the primary factor.

Now, should such flattened adhesions arise between the various abdominal viscera, one of which at least possesses mobility,—in other words, possesses the power of changing its position in relation to the neighboring organs,—whether this be by means of peristaltic movement, continuous displacement of the diaphragm, or the alternate filling and emptying of a hollow viscus, or by its own weight—in many cases the form of adhesion gradually changes, and, in some instances, rupture of the bands will result.

The blood-vessels of the adhesions also become stretched by the tension, their lumen becomes narrowed, and finally may even become completely obliterated, quite in the same fashion as the capillaries of the alveoli of the lungs do in empyema. Furthermore, from this fact, the nutrition of the adhesion naturally suffers, so that the latter becomes thin and, under certain circumstances, may completely rupture; the torn ends shrink up, and, as an ultimate result, only a thickening of the peritoneum remains; and, lastly, the formation of small cysts occurs, as has been observed clinically in a number of abdominal sections. More frequently, however, the more resistant portions possessing blood-vessels of larger calibre offer greater resistance, whereas the thinner portions disappear, as described above. From this the shred-like adhesions, which are often observed, arise.

I have just emphasized the fact that adhesions only occur when the peritoneum has been irritated mechanically, or from infection. The endothelium of the serous membrane must have lost its

smoothness, and with this is lost one of its physiologic functions, namely, the sliding property of the abdominal organs one upon the other, covered as they are by the serous membrane. Such injuries to the peritoneum may be mechanical, chemical, or bacterial in nature. These varieties of etiologic causes, as well as the extreme delicacy of the endothelium, which is readily disturbed physiologically and anatomically from the very slightest injury, easily explain the very frequent occurrence of peritoneal adhesions.

In the severe forms of general peritonitis, following perforation of the stomach or intestines, of the liver, spleen, and pelvic processes, resulting from infection of the generative organs of the female, death may occur before adhesions between the abdominal organs take place; or when the patient lives the purulent or fibropurulent exudate may become walled off, a condition usually occurring in the milder forms of peritonitis, which result principally from a continuation of an inflammatory process of one of the abdominal viscera. This also occurs in non-perforating abscess and ulcerative processes of these organs, if the primary cause is removed; and in fact this may come about in such a way that the exudate, which may be of a seropurulent or serofibrinous nature, saturates the inflamed serous surface to a greater or less extent, and instead of the layer of desquamated endothelium becoming piled up, it is absorbed. Then, in rarer cases, either the endothelium becomes regenerated or a vascular and cell proliferation of the embryonal type occurs, which later on results in tough adhesions according to the intensity of the process. This either results in a thickening of the serosa, or it represents an organized coalescence with a neighboring organ,—namely, an adhesion. An adhesive peritonitis, such as this, is far more likely to be circumscribed than diffused. Occasionally it arises from direct infection, as, for example, the entrance of bacteria through the tubes or appendix, by a dirty needle used for the withdrawal of an ascitic collection, or inattention to surgical cleanliness during abdominal section; in the majority of cases, however, at the present time, it is usually the result, as has already been mentioned, of a continuation of some inflammatory process within the abdomen. In point of fact, the agglutination of all the abdominal viscera supplied with a serous covering may arise when an inflammatory process has become established and has extended

to the surface. This very pregnant remark of Reidel may be considered in an inverse sense, all the more so since it is probable that the existing adhesions may be looked upon as a septic lymphangitis, inasmuch as by means of the lymphatics a centrifugal propagation of the inflammation to the surrounding peritoneal surfaces can arise, as has been demonstrated microscopically by Sundberg.

Here they produce peritoneal manifestations, such as redness, hyperemia, tumefaction, and exudation, especially of a fibrinous nature. One is dealing with a lymphatic propagation of the inflammation in the great abdominal lymph sac to the peritoneum. At the same time the endothelial layer is destroyed.

If the process extends over to the neighboring peritoneal surface the same condition of affairs occurs; the masses of fibrin coming from each side become agglutinated, the meshes of fibrin become organized, and the formation is complete. According to the organ in whose neighborhood a localized peritonitis occurs, one refers to the process as a perigastritis, perityphlitis, perisalpingitis, etc. In this way a number of adhesions form around the focus of inflammation, cutting off the diseased parts from the surrounding healthy structures. If, now, the focus of infection should rupture into the peritoneum no perforation occurs into the free abdominal cavity, because the adhesions on the outside of the peritoneal covering of the diseased organ have provided a barrier. A pus focus which increases in size within its adhesions stretches them and new ones continue forming at the periphery, and the larger the abscess becomes the more extensive will be the adhesions, and the farther they will advance into the peritoneal cavity. This process of walling off of the intra-abdominal inflammatory focus occurs more particularly in appendicitis, gastric ulcer, salpingitis, cholecystitis, metritis, etc., and, of course, requires a certain amount of time when the inflammation is not too acute and progressive. When the bacteria are especially virulent and in unusually large numbers, they may so disturb matters that the protecting wall does not arise, and then perforation, with propagation into the free abdominal cavity, takes place, leading to an acute general peritonitis. Fortunately this latter occurrence is clinically the exception, as the

ability of the peritoneum to wall off the inflammatory focus by means of adhesions is nature's extremely important mission.

On the other hand, the formation of adhesions often incurs great danger, because, even after the inflammatory process has completely run its course, the adhesions remain behind—often for many years after the primary affection has disappeared; and the result is that these patients suffer severe pain, or occasionally intestinal obstruction. Aside from this the adhesions frequently set up a certain characteristic train of symptoms, according to the location and type of infection. This, I desire to describe from the clinical point of view, and I shall first consider adhesions occurring in appendicitis. Their occurrence in this affection forms a rule to which I believe there are few exceptions. In the catarrhal type there is always microscopic evidence of bacteria in the wall of the appendix, accompanied by septic lymphangitis, which results in adhesion formation. The appendix at first becomes adherent to the immediate surrounding structures, and when the intensity of the inflammatory process increases there may result an exudate and abscess formation with or without perforation. Coils of small intestine become adherent at points quite distant from the diseased organ, while the colon, omentum, and parietal peritoneum may become involved.

The great advantages of the walling off of the focus of inflammation have already been emphasized. However, it must be admitted that the adhesions present certain disadvantages, because at the expiration of the acute inflammatory outbreak they may tear off or strangulate the appendix, and they certainly play an important part in the etiology of relapsing appendicitis. From this fact they restrict the peristaltic evacuation of the organ, and it now merely requires a traumatism or an indiscretion in diet, with its consequent increased peristalsis, to make the obstruction complete and with it to light up the old inflammatory process again.

Naturally the prognosis of such a relapse is favorable on account of the presence of the adhesions in direct relation to their strength and amount, and, generally speaking, it is my opinion that peritonitis does not seem to occur often in these cases, because frequently we find large abscess-pockets which have healed by granulation, and when the adhesions are extensive and the case relapses, a new

abscess must certainly develop and undergo the same regression as the first one did.

It can be readily conceived that the danger of exudative perityphlitis bursting into the free abdominal cavity must diminish with each attack, for the adhesions become further advanced in each one and thus make more complete, the walling off of the appendix as well as the pus focus arising from the organ.

Kummel has made the statement that the greatest mortality in cases of appendicitis occurs during the first attack, an assertion whose correctness has been verified in my personal experience in cases of generalized peritonitis arising during appendicitis. My experience also has brought me to the conclusion that the first attack is especially dangerous in children, and it would almost seem as though in a certain number of people, at least during the progress of years, and without any noticeable symptoms, changes arise in and around the appendix which act as a protection against perforation of the organ when it becomes inflamed.

In periappendicular abscess the adhesions allow the pus to make its exit and form around the appendix, without attaining the abdominal cavity—in all those instances in which the abscess reaches the parietal peritoneum, whether it be on the anterior abdominal wall, in the true pelvis, or in the posterior cul-de-sac. Although we are able to decrease considerably the dangers accruing from an intraperitoneal abscess by carefully walling off the general peritoneal cavity and plugging with gauze, it is always a better proceeding to drain the pocket by the vagina in the first place, when this is possible. It is still a much moot question among surgeons whether or not the appendix should be searched for and removed after incising a periappendicular abscess without opening the free abdominal cavity, but it may be said that a number of prominent German operators recommend it.

As advantageous as it may appear immediately to remove the primary cause of the trouble, it nevertheless must be admitted that this proceeding frequently carries considerable danger with it, and, for this reason, probably, most surgeons prefer the interval operation when the appendix is not within sight, or is bound down too closely by adhesions. Personally, it has always appeared to me a dangerous practice to search for the appendix after opening the

abscess-cavity and letting out the pus, for frequently the organ is bound down in a mass of adhesions, which compose a certain portion of the wall of the purulent pocket. If the appendix lies deeply hidden, the wall of the abscess will certainly be ruptured and the general peritoneal cavity will be thus infected, and then, again, very often coils of intestine are adherent to the wall and if there is much manipulation in searching for the appendix they may be easily perforated without knowledge of the operator.

Just as in the case of the appendix some portion of the intestine may become stenosed, or even entirely obstructed. This, however, as well as the pains to which adhesions may give rise, I have considered in another paper.[1] I would merely remark that symptoms produced by the adhesions may simulate a chronic appendicitis, or should they give rise to colic-like attacks one may infer that there is a recurrence of the inflammatory process in the appendix.

Then, again, the appendix may be destroyed or obliterated, and with this occurrence the true pathologic process may disappear permanently. It is a question whether or not, in those cases in which the appendix cannot be removed, the continuation of the subjective symptoms results from the pathologic changes arising in the appendix or are due to the adhesions, and cannot be determined until a primary or secondary operation is undertaken. However, in reality, this is of little import, because the operation, even in the case of adhesions, will result in relief and cause the symptoms entirely to subside. This result, however, is not always attained with certainty, especially when extensive adhesions exist (as one meets with more especially in instances of frequently-recurring attacks of appendicitis), because, at the present time, we are unacquainted with any certain method to prevent an occurrence of adhesions or their removal, and consequently, after they have been broken up they are prone to recur, although it must be admitted that they are less bothersome than before the operation was undertaken.

It, therefore, becomes evident that an early operation is most advisable, aside from the fact that the patient is free from further trouble, because by operation the formation of extensive adhesions and their ultimate consequences are prevented.

[1] Abdominal Pain from Adhesions. The Clinical Journal, London, May, 1905.

Very serious, or even dangerous, adhesions from appendicitis may involve the female genitalia, aside from the fact that they may give rise to pain if pregnancy takes place, on account of the increasing size of the uterus, or that during labor the appendix may become ruptured by traction resulting from the adhesions binding it to the uterus, which may result in the lighting up of an old and latent inflammatory process. Such instances are, at the present time, not infrequently met with, since the pathology of appendicitis has been thoroughly elucidated.

We now come to the consideration of adhesions arising in gastric ulcer. When this pathologic process has extended so deeply that it has encroached upon the serosa, an adhesion usually results between it and the adjacent peritoneal surface. These adhesions are most prone to occur between the stomach and the liver, the pancreas and omentum. On the other hand, the anterior wall of the stomach has much less tendency to adhere with the parietal peritoneum to which it is adjacent, but this fact is probably due to the continued motion of the anterior abdominal wall and the stomach, which, during respiration, move in opposite directions.

Now, then, in other portions of the stomach a threatened perforation is frequently prevented by adhesion formation, but this does not occur on the anterior aspect of the organ, and, for this reason, a complete perforation is more apt to arise here than in other regions of the organ, some authorities placing it as high as 80 per cent. of all gastric perforations.

Fortunately, only about 5 per cent. of all gastric ulcers are located in this unfavorable place, because they are principally localized on the posterior wall of the lesser curvature and pyloric region. When the ulcer has perforated the wall of the stomach it occasionally progresses through the tough adhesions and thus reaches the viscera with which the stomach is adherent, the ulcerative process progressing directly through the adhesions. Such an ulcer, which Schwarz has aptly termed "penetrating," erodes, according to its position, the liver, spleen, pancreas, and less frequently the anterior abdominal wall. In some rare instances the ulcer perforates an intestinal coil which is adherent with the stomach; most frequently perforation of the transverse colon, or a gastro-intestinal fistula, thus results.

If perforation of the stomach-wall takes place rapidly before any firm adhesions have formed, the result is a sudden outpouring of the stomach contents into the abdominal cavity, followed by all its disastrous results and well-known complications.

In other cases adhesions have already formed around the spot of perforation, so that an outpouring of the stomach contents into the general peritoneal cavity cannot take place, but the adhesions may be so loose that they are incapable of resisting sufficiently the occurrence of a local, or progressive, peritonitis. Either an absorption of the encapsulated effusion will result, leaving behind broad and tough bands of adhesion, or an abscess develops, which, later on, may perforate into the free abdominal cavity, or into the neighboring organs.

Most of the cases of subphrenic abscess arising on the left side are usually the result of a subacute perigastritis. The so-called penetrating ulcer of the anterior wall of the stomach will readily give the impression of a tumor when the region is palpated, probably mainly on account of the inflammatory infiltration situated around the base of the ulcer directly under the abdominal wall. This pseudoneoplasm readily gives rise to an erroneous diagnosis being made, usually carcinoma of the stomach or else a malignant tumor of the abdominal wall; and indeed this is especially prone to occur on account of the increasing intensity of the pain and long duration of the disease, the general health suffering practically as much as in cases of carcinoma, the patient presenting marked evidence of cachexia. Then, again, extremely severe pain, localized in the region of the tumefaction, and occurring more especially after the ingestion of food, closely resembles the pain complained of in gastric carcinoma. In a case of ulcer there are two causes which may give rise to these pains: first, they may be due to the extension in depth of the ulcerative process; or, again, they may depend upon the adhesions resulting from the process in the stomach. In the former case, which does not admit of internal therapy, resection of that portion of the stomach which is involved by the ulcer represents without doubt the most radical method; it removes the focus of disease and at the same time does away with the adhesions. However, it must be admitted that such an operation is only permissible when the site of the ulcer is favorable for

its easy execution,—in other words, when it is located in the anterior wall of the organ. In other cases the mere breaking down of the adhesion is sufficient to do away with all symptoms, while in others, those in which the ulcer is located in a position not easily accessible, a cure may be brought about by gastro-enterostomy.

There are a few other disturbances in the motility of the stomach which should be mentioned and which occasionally may be produced by adhesions. I refer especially to pyloric stenosis. As a rule, contraction of a cicatrix from an ulcer is the etiologic factor of a non-malignant stenosis; but, in some rare cases adhesions set up by an ulcer located in the pylorus may in themselves narrow the lumen, or make a preëxisting stenosis still more marked symptomatically.

Adhesions arising after pericholecystitis also frequently have the same results. The narrowing results from the bending due to the high fixation of the pylorus. Breaking down the adhesions will, under these circumstances, relieve the pyloric stenosis, but as one is never sure that a recurrence of the adhesions may not take place, it would seem that in these cases gastro-enterostomy is also indicated.

In cholecystitis, when there is an intense and severe infection of the gall-bladder, with swollen walls resulting from an inflammatory exudate, or when infiltrated with pus, in consequence of this ulceration and necrosis of the mucosa, an extension of the inflammatory process reaching to the serosa inevitably results, with a pericholecystitis and adhesion formation. From this a perforation which might occur into the free abdominal cavity is usually prevented and instead a fistula occasionally arises between the gall-bladder and some adherent hollow viscus. As a result of presure produced by a gall-stone, necrosis, perforation, and a fistula may result. Thus a calculus located in the duct and impacted there may attain the interior of the intestine by gradual adhesion of the duct with the wall of the gut. In quite a similar way calculi will sometimes come directly from the gall-badder into the intestine through a fistula existing between the colon and the fundus of the organ.

Another method of recovery from cholelithiasis is brought about by a fistula arising between the gall-bladder and the abdominal wall. In these cases the external opening is usually found in

the neighborhood of the umbilicus, the stones being finally expelled through this channel. In very infrequent cases, perforation of the gall-bladder into the stomach, bladder, or pelvis, may take place.

Now, if even all the stones may have been evacuated, thus doing away with the causa nocens, the old colicky pains, and occasionally also the icterus, still remain on account of the presence of adhesions which press upon and obstruct the gall-bladder or the ducts, with consequent retention and infection. In some instances in which this condition of affairs has been found, breaking down the adhesions and covering the raw surfaces with peritoneum have resulted in recovery. In these cases typical attacks of colic and severe icterus existed as a result of obstruction to the choledochus from adhesions. I have already pointed out that stenosis of the pylorus may arise from an extension of a pericholecystitis and that it requires surgical treatment. Perimetritis is an extremely frequent complication in diseases of the internal female genitalia. This is due to the fact that the causes of infection do not always extend to the serosa first by the way of the lymphatics, but penetrate directly by way of the tubes to the peritoneal cavity.

The severe inflammatory processes of the pelvic peritoneum, occurring mainly during the puerperium, usually of a purulent character, usually become generalized and result in death; but, on the other hand, the milder forms with fibrinous, serofibrinous, or fibrinopurulent exudate, usually remain localized and after resorption of the exudate lead to adhesions. This pelvic peritonitis with adhesions occurs more especially in cases of parametritis, generally of puerperal origin, in gonorrhœa of the adnexa, or a dirty intra-uterine operation. The adhesion formation shows the most gradual anatomic differences; sometimes after a slight catarrh of the tubes a few delicate strands, or some firmer adhesions, will be found; but soon, however, the uterus, adnexa, single coils of intestine, or the tip of the omentum, become imbedded in thick bands. Between these all stages may be met with.

Imbedded in the adhesions remains of the exudate are frequently found, or even purulent foci, which either become slowly absorbed or may rupture through the adhesions at any point, into either the free abdominal cavity, bladder, vagina, or intestine. The primary inflammation, however, need not necessarily start from the

genitalia, because ulcerative processes of the rectum may give rise to inflammatory adhesions of the pelvic peritoneum.

Aside from the severe manifestations resulting, and the disturbances of the intestinal canal, the greatest serious import of perimetritic adhesions resides in the fact that from their presence the pelvic organs are changed as to their position, become distorted and bound down, resulting in numerous circulatory and functional disturbances. The conditions for recovery become considerably worse and the patients remain invalids for their entire life. Although the inflammatory process may have entirely subsided, in most cases as a result of a long-standing malposition of the uterus and adnexa from the adhesions, sterility results, or at any rate renders conception a difficult matter.

Such a blocking up or bending of the tubes may be a danger to life when it interferes with the passage of the fertile ovum and leads to extra-uterine pregnancy.

Retro-uterine hematocele occurring from rupture of a tubal gestation very often becomes walled off by adhesions, although, as I shall point out later, the blood in itself does not give rise to these adhesions. They most probably arise from the inflammatory process resulting from infection of intestinal origin: the walls of the gut become permeable to bacteria from the resulting pressure upon them, or from chronic perimetritis which in itself is due to an occlusion of the tubes.

In considering adhesions arising in chronic peritonitis, the tuberculous form stands preëminent. It goes without saying that only the adhesive type of this disease is of interest, although it should be recalled that this is not infrequently combined with the ascitic type. The formation of adhesions upon the diseased peritoneal surface, as in acute inflammatory processes, appears to be due to the efforts of the organism to wall off the foci of disease. This, however, in no way prevents the eruptions of tubercle on the membranes after a more or less long lapse of time.

If intestinal tuberculosis arises at the same time, the adhesions not infrequently prevent perforation of tuberculous ulcers into the free abdominal cavity, and by inclosing them a mixed infection from intestinal bacteria usually results in a purulent exudate. The viscera frequently become agglutinated very extensively and form

a practically unrecognizable mass, and, on account of this, palpation will readily deceive one, because a tumor is thus formed and may be mistaken for some intra-abdominal neoplasm. Then again the greatly thickened omentum becomes, so to speak, rolled up, and may also be mistaken for a growth, frequently of the liver, because these organs are occasionally adherent to each other.

As characteristic of pseudo-tumors resulting from the matting together of the intestines, a fairly rapid change, both in volume and consistency, may be observed if frequent examinations be made.

According to the way in which the adhesions form, the fluid exudate, which is so often present among them, loses its free mobility and becomes occasionally walled off in such a manner that it can easily be mistaken for cystic growths, as, for example, an ovarian cyst, or those due to echinococcus infection. It goes without saying that these pseudo-tumors do not have that regular and distinct outline such as is found in a true cystic tumor. Beside the general tuberculous peritonitis a local tuberculous peritonitis also arises; this is generally dependent upon intestinal tuberculosis, more rarely upon tuberculosis of the mesentery, retroperitoneal glands, or the spinal column. More frequently, it is found coincident with the favorite site of intestinal ulcers of a tuberculous nature in the ileocecal region. At this point what may be termed a tuberculous ileocecal tumor develops, first as the result of thickening of the walls of the affected portions of the intestine, and also from adhesions with the surrounding parts; thus arises tuberculoma cæci, which is very often difficult to differentiate clinically from a carcinoma or a periappendicular tumefaction.

There is another form of chronic adhesive peritonitis which is not tuberculous, and has been described as chronic idiopathic peritonitis, in which thickening of the peritoneum results with adhesion and contraction, the latter especially arising in the peritoneal covering of the mesentery. This still indefinite disease, so far as its etiology is concerned, is important, less so on account of the formation of adhesions, which show nothing of special import, but on account of the shrinking and cicatricial formation, which may ultimately lead to stenosis and kinking of the intestine and result in volvulus.

Considering now adhesions arising from the presence of neoplasms, it may be said that primary thickening of the peritoneum and tumors lying in the subserous region of organs covered by peritoneum do not, generally speaking, give rise to adhesions. However, such subperitoneal growths as, for instance, ovarian cysts or uterine myoma, can attain an enormous size and extend far up into the abdominal cavity without giving rise to a single adhesion. Occasionally, however, these tumors may become adherent to the abdominal wall, the omentum, the small intestine, etc., so that much difficulty is experienced in their removal. I am of the opinion that not infrequently an irritation is set up by pressure from corsets and skirts on the tumor which is in contact with the abdominal wall from which adhesions result. Then again an aseptic traumatism may result in injury to both layers of endothelium of the peritoneum, so that adhesions develop between the tumor and the surrounding organs or abdominal wall.

Another explanation may also be given; similarly to the striæ arising during pregnancy in the skin of the abdomen, injuries to the peritoneum forming the capsule of rapidly-growing tumors may occur in the form of minute ruptures and these become loci minoris resistentiæ. Now, as I shall point out later, an aseptic injury of the peritoneal surface does not lead to adhesions; but the above-mentioned minute ruptures, if they become infected, will be favorable for adhesion formation. The infection has been supposed to arise in this manner, namely, that the growth, by the pressure it exercises on the intestine, causes the wall of the gut to become permeable for the intestinal bacteria. Boenneckon some time ago demonstrated that bacteriologically the fluid in a hernial sac, which had been walled off for only twenty-four hours, contained organisms, although no obvious changes in the texture of the wall of the gut were required to make the latter permeable to bacteria, and that a fairly severe venous stasis, a marked serous saturation of the tissue, suffices to permit of the penetration of bacteria into the intestinal wall through which they progress outwardly. Certain authorities maintain that the pressure of the growth might play the same part as the tight hernial ring, and they have been upheld in this assumption by the investigations carried out by d'Anna, who demonstrated Bacterium coli in the peritoneal fluid resulting from the presence of

abdominal growths. The minute peritoneal ruptures may also be produced by slight traumatism due to tight clothing, etc. Which of these explanations is the most plausible I am not prepared to decide, but it might be admitted that both of them are etiologic factors for adhesion formation. The adhesions arising in gastric ulcer or carcinoma of the intestine, have the same infectious origin as in other ulcerative processes of the stomach and intestine.

Concerning the manner of development of adhesions in hernia it may be said that they have the same causes, such as slight traumatism and, above all, infection. The exposed site of the contents of a hernia, the usually narrow space given by the external ring, frequently results in mechanical injuries and injurious pressure, which make the intestinal wall permeable to bacteria. This is, above all, apt to arise in unskilful and rough reduction.

As a result of this, slight injuries are produced and the hernial ring and sac become thickened and lose their elasticity. From this fact the hernia loses its ability of being easily reduced, and, if it should not be replaced within the abdomen for a certain length of time, adhesions of the intestinal coils with the sac and a tab of omentum, frequently arise, and from a reducible hernia an irreducible one results.

Aside from the other symptoms of hernia various sorts of digestive disturbances, which are characteristic of an irreducible rupture, arise, such as sharp colicky pains, more especially caused by the presence of adhesions dragging on the gut. The principal danger of irreducible hernia is, as is well known, strangulation, which, above all, develops in the form of stercoracious incarceration.

The operation relief for these strangulated hernias frequently becomes extremely complicated for the reason that a safe isolation of the intestine from the adherent sac is made an extremely difficult matter on account of the soft bluish black condition resulting from blood and lymph stasis. The result of all this has been that not infrequently the gut has ruptured on account of its very soft and friable consistency. Recently, however, a method has been employed which avoids this dangerous occurrence and has been termed laparo-herniotomy. This technic, which also protects against infection of the abdominal cavity, as well as hemorrhage, has the great advantage that it is much simpler and easier than the classical abdominal

incision. It may be briefly described as follows: The abdominal cavity is opened above the strangulation, and through this incision the hernial ring and sac are split open from above,—in other words, just the reverse of the proceeding ordinarily employed. The surgeon then follows the parts from this point where they are normal to that where they have undergone the pathologic changes arising from the strangulation and which make reduction difficult. In this way, if due amount of care be exercised, the intestine can be safely loosened from its adhesions without rupturing it. In those hernias which have no intestine for a contents, as, for instance, hernia of the linea alba, which usually contains only omentum and in which no infection has occurred, the adhesions met with are perhaps the result of the tight squeezing of the neck of the sac.

Contusions of the abdomen which do not result in severe internal injury, such as rupture of the larger vessels or perforation of the stomach or intestine, frequently ultimately result in severe gastro-intestinal disturbances, principally characterized by pain, severe colic, vomiting, attacks of intestinal obstruction or even ileus, all these resulting from the formation of adhesions and coming on after a considerable lapse of time after the receipt of the injury. A large number of surgeons have described such cases in detail. The clinical appearances will be discussed later and I would at this point consider only the question of the development of adhesions arising from traumatism.

Among those injuries of a mild character, which I am only here considering, as a result of contusion of the abdomen, superficial rupture of the liver, spleen, stomach, intestine, and bladder may arise; in the latter organ the peritoneum or the mucous surface may be injured from the introduction of instruments, etc. Then, again, crushing of the various organs resulting in the formation of hematoma and partial necrosis may result.

Now when two injured serous surfaces lie directly opposite each other an adhesion easily arises. This type of adhesion formation is probably rare, as they can only occur from a mere coincidence, and for this reason Vogel assumed that they develop from organization of blood in a purely aseptic way. This, however, is very improbable because, as I shall attempt to show later, aseptic blood, even if it originate from peritoneal injuries, becomes obsorbed without leav-

ing any trace. The most probable explanation is that infection occurs and the adhesions result from a slight local peritonitis. Every ruptured wound in the layers of the intestinal walls forms an entrance through which the intestinal bacteria may make their way and, according to the anatomic course followed by the lymphatic and blood-vessels of the intestine, their colonization near the peritoneal covering is inevitable, although the solution of continuity may not extend to the deeper strata.

Likewise it can easily be understood that points of the stomach or intestinal walls which have been crushed, resulting in slight rents of the serous membrane and subserous hematoma must permit of a centrifugal microbic emigration quite similar to that occurring in strangulated hernia.

Lastly, although infrequently, indeed, a necrosis of the mucous membrane and a traumatic ulcer develop and with them, similar to other ulcerative processes, the formation of adhesion can be brought about.

From all this it will be seen that there are many possibilities of infection, and should a hemorrhage occur at the same time, the bacteria are offered the best opportunity for their development in this culture media. From all this it can be easily explained that occasionally more extensive adhesions develop than would have otherwise from the site of primary injury. Injuries to the gall-bladder and ducts, in which bile penetrates into the peritoneal cavity, result in the formation of adhesions for the simple reason that the fluid is chemically different from that of the peritoneum and destroys the endothelium.

Thanks to the ability possessed by the peritoneum rapidly to form adhesions it has become possible to attack foci of infection lying intraperitoneally, as well as abscess of the liver and spleen, empyema of the gall-bladder, tubes, and appendix, in such a way that by establishing an isolation of the focus of infection by adhesions the pockets may be opened and evacuated with safety.

In just this way adhesions are useful in all operations in which the lumen of the digestive tract is opened and, for example, intestinal réactions and anastomoses can be undertaken on account of the rapid development of adhesions between the openings made in the gut or stomach.

Adhesions of the omentum, mesentery, and intestines are produced around the point of suture, whether the cause be manipulation of the viscera, resulting in mechanical damage to the peritoneum, or small masses of the infectious stomach or intestinal contents reaching the surrounding border of the wound, which practically can never be prevented during operation. Such adhesions are advantageous in that they have no real action on the healing process; they fix the intestine in a certain position and, so to speak, splint it, which is exceedingly important in all processes of repair. They finally help by covering over the line of suture and prevent a solution in continuity. That these adhesions in septic laparotomies have their dangers is proved by many instances of intestinal occlusion following resection of the intestine, etc.

Differing from these adhesions, which, although they occasionally give rise to unpleasant complications, are certainly nevertheless of service to the surgeon, are peritoneal adhesions arising after aseptic laparotomies, such as the radical cure of hernia, removal of uterine growths, etc., which appear practically unavoidable, according to the way of thinking of the majority of surgeons. These form unfortunate conditions, which, however, in a great majority of cases are without harm, but, in some few abdominal sections they cause the intervention to be partially or entirely without result, on account of the occurrence of ileus or abdominal pain.

Shortly after it was recognized that these unfortunate occurrences were due to adhesions, an attempt was made to ascertain the conditions under which adhesions were produced, in order, if possible, to do away with their formation after abdominal section. That adhesions do occur as a result of infection, as well as between two serous surfaces which are in contact, needs no further discussion, but again the question comes up as to whether or not an adhesion may develop when only a single raw surface exists, as, for example, when the pedicle after ovariotomy comes in contact with healthy peritoneum.

In order to settle this question Sänger undertook some animal experiments, resecting a large bit of peritoneum and abdominal muscle in rabbits. By this means he obtained firm and extensive intestinal adhesions and from this he drew the conclusion that, after

a defect in the peritoneum, new endothelium is not formed and that the endothelial covering of the intestine suffices to prevent adhesions with the abdominal wall. On the other hand, von Dombowsky reached absolutely different results for, in several of his experiments, he manipulated the peritoneum with a toothbrush until the lustre of the serous membrane was lost and its surface became rough. In reopening the abdomen eight days later, no adhesions were found, and the endothelial covering of the injured peritoneum had become regenerated. He obtained exactly the same conditions by removing two oval pieces of peritoneum, including the fascia lying under it, and a week later he found the resected parts glistening and smooth. From these experiments he concluded that not only superficial endothelial injuries, but also deeper defects and wounds of the healthy peritoneum, healed perfectly without giving rise to adhesion formation.

The experiments of Kelterborn show the same condition. He removed the serosa from the small intestine and from the abdominal wall with a curette, and after three weeks no adhesions had arisen and the points scraped were glistening and smooth. In resection of the parietal peritoneum, partly with and partly without the underlying fascia, no adhesions resulted. Duschinsky comes to the conclusion, from his experiments, that simple peritoneal defects of a fairly good size and depth never result in adhesion, and Thomson also found that superficial peritoneal defects undergo repair without any untoward results; while, on the other hand, the omentum and intestinal adhesions arising without infection, that is to say, in sterile lapartomy wounds, as well as in abdominal muscle injuries on the anterior abdominal wall, almost constantly arise.

Vogel produced endothelial disturbances on the parietal and visceral peritoneum by rubbing them with dry gauze. No adhesions resulted when the irritation was applied some distance from the abdominal incision, while, on the other hand, they were present when the irritation was carried as far as the borders of the latter. Adhesions formed when the peritoneum was irritated by incisions made near the abdominal wound by the use of the curette. The production of adhesions in the latter case was described by Vogel as follows: The various injuries to the peritoneum converge toward one point; the rubbing and grating, the abdominal in-

cision, whose edges probably are never so exactly coapted that small raw surfaces do not exist on the intra-abdominal peritoneal aspect, the stitch-holes, as well as portions of the catgut lying intra-abdominally—and, lastly, infection, probably can explain the conditions already mentioned.

Experiments in which the cautery was used show the following results. First, we have the case reported by Kustner, in which he removed a very adherent ovarian tumor and sectioned the adhesions with the thermocautery. Fourteen months later the abdomen was opened again on account of the abdominal tumor and all adhesions had disappeared, the peritoneum being entirely smooth. The results of experiments undertaken are briefly as follows: Dembowsky produced necrobiotic changes on the parietal peritoneum with the cautery and later on found adhesions with the omentum and stomach. Kelterborn was unable to obtain adhesions by the use of the cautery on the peritoneum of the intestine, stomach, bladder, liver, and parietal peritoneum. Maslowsky, as well as Spiegelberg and Waldeyer, on the other hand, produced adhesions in their experiments. Ducchinsky says that burned surfaces of the peritoneum do not in most instances result in adhesions, while Vogel found that deep burns do not lead to adhesion formation, although superficial ones do.

The results of the various investigators are, as is seen, quite different and there is good reason for this. As I have already pointed out, the peritoneum reacts to slight injuries due to infection, and chemical or physical insults, resulting in adhesions. To exclude those injuries in experiments in which the influence may be studied in only a single case, is extremely difficult and perhaps even impossible. Ultimate results of these experiments have, therefore, only a very slight value, aside from the fact that the results of animal experimentation cannot be carried out in man. The negative experiments appear to prove that, at the most, no adhesions resulted, whereas in experiments giving rise to a positive result, it may be rightly concluded that some other unintentional injury which passed by unnoticed, was the factor in their formation. Then, again, in these experiments, it must be taken for granted that single peritoneal defects resulting from the cautery do not lead to adhesions. As a matter of fact, a large majority of writers have come

to this conclusion, and, as a further basis for this assumption, Kelterborn recalls the analogy in a normal female during sexual life in whom intraperitoneal epithelial injuries arise. He emphasizes the point that, during rupture of the Graafian follicle, a raw intra-abdominal peritoneal surface arises which undergoes repair, leaving behind a cicatrix. This scar formation does not give rise to adhesions. The explanation of this is very simple; a fresh, clean and aseptic wound will undergo normal repair in any tissue of the body and consequently in the abdominal cavity no adhesions will arise between the neighboring organs and the ovary. From all this I believe a justifiable conclusion is that an aseptic defect at one point of the peritoneum will never lead to adhesion formation. Whether the injury be deep or superficial, simple or complicated, is of no importance whatsoever, for one cannot readily conceive why simple incision and peritoneal defects do not damage the neighboring peritoneal surface and lead to the formation of adhesions, while, on the other hand, severer injuries show that nothing results.

It is an entirely different matter if, during an operation, infection of the injured tissue follows. In this case the adjacent serous surfaces are so much injured by bacteria and their toxins that the two become adapted for adhesion formation. There then is a question of two pathologic defects. It may be that the results of the experiments are due to a great extent to an unintentional infection of the peritoneum, which only too often is supposed to occur; and it is found that aseptic cauterization does not lead to the formation of adhesions, but, on the other hand, adhesions always occur when an intentional or unintentional infection is produced, as shown by cultures and the microscope. An infectious process need not give any great signs of its presence; perhaps merely a slight elevation of temperature. Brink took this factor into consideration in his experiments on absorption fever. Primary union may not be otherwise disturbed. The peritoneum with its great bactericidal faculties readily becomes master of the infectious process. The bacteria at first can develop in the locus minoris resistentiæ of the peritoneal wound surface or in the blood-clot, sufficiently to produce changes leading to the formation of adhesions. Infections of this kind, in otherwise perfectly aseptic laparotomies, are usually the result of

the operator's hands. It is only quite recently that by the use of rubber gloves infection may be avoided, for I think I have conclusively demonstrated elsewhere that absolute asepsis of the hands is an absolute impossibility,[2] a conclusion that has been reached by a large number of other investigators during the last decade.

The abdominal wound itself is readily predisposed to infection; therefore adhesions arising from the suture of the abdominal wound, if infected, a thing which is very prone to occur from the operator's hands if not protected by rubber gloves, will give rise to adhesions. A sterile suture in itself will in all probability very rarely give rise to adhesion formation, and, when it does so, an infection is at the bottom of the process. Tenbrinck found adhesions with the line of abdominal suture in ten of his experiments and inoculation was positive in all excepting two cases, in which bacteriologic examination of the abdominal suture was negative. He consequently draws the conclusion that without infection no adhesion will arise.

Just as long as we remain ignorant of an absolutely reliable method for hand disinfection, these infections cannot be excluded with any amount of certainty, but it is indeed possible to avoid inoculation with highly virulent bacteria, which the peritoneum would not have the power of overcoming.

The other injuries which I shall soon discuss, and which give rise to adhesions, can very easily be avoided with our modern aseptic technic. Steffens, in an excellent work relative to secondary laparotomy, has shown that in nineteen cases healing of the abdominal incision in the first laparotomy occurred with an elevation of the temperature. In every one of these nineteen cases adhesions were found at the second operation. In fourteen cases which healed without any rise in temperature adhesions were also found, and of these, twelve would naturally have resulted in adhesion formation, because they were instances of resection of the stomach, intestine, appendicitis, tuberculous peritonitis, retroperitoneal abscess, cholelithiasis, and carcinomatous metastases; in two cases he was dealing with the removal of very adherent benign growths from which multiple wound surfaces resulted. In only three cases, one of which

[2] Cumston and Rickerts, Hand Sterilization, Boston Medical and Surgical Journal, 1905.

had been submitted to a laparotomy three times, were no adhesions found and repair took place without any elevation of the temperature. Steffens concludes from the above study that the formation of adhesions after laparotomy is due to infection and that in consequence of an imperfect asepsis in the wound more or less extensive and infectious peritonitis arises; but having a local character this does not lead to a general infection of the patient, but adhesions do arise.

Of those injuries which may also give rise to adhesion formation after laparotomy must also be mentioned those fluids which are chemically different in composition from that of the peritoneal fluid, the influence of the air from its drying effect and loss of heat, and finally foreign bodies.

Chemical solutions, especially those employed when antiseptic treatment was in vogue, such as carbolic acid, sublimate, boracic acid, etc., rob the peritoneal surface of its endothelium and adhesions naturally develop at those points which have come in contact with the irritating fluid. If, on the other hand, the chemical agent simply injures the peritoneum at one spot the latter acts as if it had been cauterized and no adhesions will result. Vogel treated the peritoneum with nitrate of silver in four of his experiments and never obtained adhesions. Iodoform appears to have a peculiar action, because in four experiments carried out by Dembowsky and in two of Vogel's, small amounts of iodoform were apparently absorbed without leading to the formation of adhesions. Nevertheless one may assume that in some way greater amounts of iodoform acting as a foreign body, may cause adhesions not only in the form of a chemical irritant, but animal experimentation appears to disprove it. Pure, undecomposed bile and urine also belong to the fluids which irritate and injure the peritoneum; they produce an aseptic adhesive peritonitis.

In reality the blood does not give rise to any adhesions and becomes absorbed without leaving any trace of its presence. Under normal conditions this occurs each month when ovulation and menstruation take place when small quantities of blood from a ruptured follicle escapes into the peritoneal cavity. However, Gersuny is of the opinion that adhesions arising around the female genitalia, which he has described as characteristic, developed partly

in his cases (thirteen out of twenty-four), apparently without any inflammatory process having been present, and were due to the profuse hemorrhage during menstruation and ovulation in which the blood was only partially absorbed, the remainder becoming organized with resulting adhesions. He comes to this conclusion because, in the thirteen cases referred to, he could not find in the patient's history, or clinically, any evidence of a previously existing inflammatory process. In reality this does not prove very much, for the inflammation may have been so slight that it caused no symptoms, or very mild ones, which were not noticed by the patients.

It is not of unusual occurrence to meet with cases in which clinically and from the history of the case, one cannot explain the presence of adhesions found. Haidenhaim has reported eight instances of intestinal occlusion produced by bands of adhesion, and in only two could the cause of the chronic adhesive peritonitis be discovered. Von Dembowsky and Vogel took up the question experimentally as to whether or not intra-abdominal hemorrhage would result in adhesion formation. The former, in his experiments, cut a mesenteric artery and allowed the mesentery to fill up with blood, which immediately coagulated, after which he ligated the vessel. A week later the blood was found to have become absorbed without leaving any adhesions, and in only one instance was the injured spot found adherent with the omentum to a small extent.

Vogel undertook the same experiments in four animals and each time he was able to obtain adhesions which were more extensive than the injured spot. In one out of five experiments he allowed the blood which he obtained by cutting one of the small arteries in the muscles of the abdominal wall to leak into the abdomen. Absolute absorption resulted without any adhesion formation. This author concluded from these experiments that the blood in itself does not act like a foreign body by irritating the peritoneum for adhesion formation; if it originates from an intraperitoneal injury it coagulates at the point injured in the same manner as in a blood-vessel with an injured intima and is then the cause of extensive adhesions. Blood coming from without and leaking into an absolutely intact peritoneal cavity, becomes absorbed without any adhe-

sion formation resulting. Injuries to a single spot of the peritoneum do not, as has already been pointed out, produce adhesion formation and this statement seems to be fairly well proved by the physiologic process of menstruation and the three experiments undertaken by Dembowsky already alluded to. Then again Vogel's five experiments apparently did not deal with a completely intact peritoneal cavity, because the abdominal suture in itself represents an intraperitoneal traumatism and Vogel himself admits that the edges of the incision are never so accurately adapted as to prevent small raw peritoneal surfaces existing.

The question arises as to how blood may reach an absolutely intact abdominal cavity, because an injury must always have preceded the advent of the hemorrhage. It is probable that, on account of its specific gravity, the blood will immediately come in contact with the abdominal incision, because animals never assume the recumbent position and very soon after operation are running about. Now if, in spite of this, Vogel found adhesions in four of his animals at the site of the injury, it becomes evident that other factors must have been present to cause this result. For instance, the ligatures placed on the injured vessels were quite sufficient, because it is generally known that they will occasionally result in adhesion formation, and this may also account for the slight adhesions found in one experiment by Von Dembowsky.

If now, however, adhesions between two injured peritoneal surfaces in contact with each other take place with coagulated blood in their vicinity, the process will be limited to the point of injury, because the coagulated blood is not inclined to have any injurious influence on the normal peritoneum surrounding the defect, as has been shown by Dembowsky's experiments, nor has the connective tissue which has become adherent to the coagulum the power of breaking through the endothelial layer; otherwise all single intraperitoneal lesions, as well as the physiologic ones arising in the ovary, would naturally lead to adhesion formation. The coagulated blood, therefore, has no influence whatsoever upon the extent of the adhesive process; the latter, however, depends upon the firmness and thickness of the clot, because the larger the clot the greater will be the amount of connective tissue replacing it, which by contraction will give rise to tougher adhesions.

If, however, the layer of adhesion is so dense that the granulation tissue springing up from both raw surfaces has sufficient time to meet and become agglutinated, it is consequently quite conceivable that in the meantime, the endothelium would make its way from the normal tissue over to the granulating surface for which not much time is required, as is proven by the experiments of Kelterborn and Dembowsky; so that both raw surfaces, although they may be opposite each other, are soon covered with their endothelial coating and no adhesions can arise. In this manner blood might occasionally have power to prevent adhesion formation, and in those cases in which infection occurs simultaneously with hemorrhage, adhesions naturally result and are more or less extensive, because the bacteria find an excellent culture media for further development in the blood exudate.

If the peritoneum is exposed for some time to the air or to contact with hydrophile substances, as, for instance, dry gauze sponges, it loses the layer of fluid covering from evaporation or by direct absorption; the endothelium suffers from this, and, as has been experimentally proved, adhesions result. There is no doubt in my mind that prolonged exposure to the air injures the peritoneal serosa, that the superficial layer of endothelial cells die, and that this exposure is of etiologic importance in inflammatory processes arising between living and dead serosa, since: (1) if two serous surfaces which have undergone the above-mentioned changes are closely approximated for some little time, adhesions form between them; (2) if contact is not of sufficiently long duration all that takes place is a thickening of the serous surfaces. Adhesion formation between normal serous membrane on one hand, and an altered serous surface on the other, is no more apt to arise in one case than in the other.

In this process other conditions of the atmosphere of a chemical or physical nature do not need to participate even though a simultaneous lowering of temperature, in consequence of vascular contraction and the resulting lowered nutritive conditions, naturally favors injury of the peritoneum. Besides all this, Walthard still further proved that the danger of an infection in a dried serous membrane is considerably greater than when the latter retains its moisture, which, *a priori,* is obvious, since, on account of the drying

and necrobiotic process of the endothelium the bactericidal faculties of the peritoneum disappear, while, at the same time, a most suitable culture media for the growth of bacteria results. The experiments of Graser already referred to verify those undertaken by Walthard. He found these disturbances leading to the formation of fibrin appeared on the peritoneum after he had exposed this membrane.

Sterile foreign bodies will give rise to adhesions in the peritoneal cavity when they exert a certain amount of irritation on the serous membrane. The type of irritation probably consists in mechanical injuries to the serosa when otherwise indifferent foreign bodies are present in the abdominal cavity. The development of adhesions occurs in such a way that at first a fibrinous exudate develops around the foreign body, which gradually becomes organized by adhesion between the organized products. Foreign bodies capable of a slow absorption become fragmented and absorbed, but to injure the peritoneum the foreign body must be of a certain size. For instance, ligatures may give rise to adhesions, but, as a general rule, it seems that these are walled off without adhesion formation. Surgery makes use of the latter process in the case of foreign bodies when plugging the abdominal cavity with gauze is resorted to in order to make a passage through adhesions from within the abdominal cavity outward from an inflammatory focus, and thus allows the escape of infectious secretions without danger of giving rise to a generalized peritonitis.

In summing up the process of adhesion formation we may conclude as follows: Adhesions arise when two serous surfaces, pathologically changed, are in contact with each other for a certain length of time. The pathologic changes can be produced by infectious, chemical, and physical lesions, and irritation from foreign bodies. When only one peritoneal surface is the seat of some pathologic change, it will only result in adhesions if it secondarily injures the adjacent serous surface, for instance, by infection or mechanical irritation, perhaps from a ligature pressing upon it, in which case the latter acts as an irritating foreign body. Infection, however, is the principal cause for the development of adhesions, although in certain cases they may be of traumatic origin, due to the development of neoplasms or pressure in cases of hernia. Aseptic peri-

toneal defects, as well as the presence of aseptic blood, does not result in adhesions.

For the production of adhesions there is one more point of great importance which has been referred to several times, namely, that although all necessary conditions for adhesion formation may be present, this may not result in those cases in which both injured serous surfaces are not in contact for a sufficient length of time to allow them to become agglutinated to each other, when, in other words, there is a constant change in their relative positions taking place. The movements of respiration, causing the displacement of the diaphragm, for instance, is the reason why adhesions of the anterior stomach-wall with the parietal peritoneum rarely arise. Then again the position of the hollow viscera changes frequently from repletion and emptying, and, above all, from peristalsis in the case of the intestine. This is a potent preventive factor, especially in those instances in which two points tending toward adhesion with each other are small, because under these circumstances, contact is more easily prevented. On the other hand, adhesion formation is favored when immobility of the intestine is produced, which, for example, always occurs immediately after laparotomies from reflex paralysis of peristalsis.

HEMORRHAGIC DIATHESIS COMPLICATING SURGICAL WORK

BY FREDERIC GRIFFITH, M.D.

Fellow of the Academy of Medicine of New York

The comparative rarity of hemophilia in part excuses the surgeon when in the midst or after the completion of an operation it suddenly dawns upon him that the patient is a "bleeder." Unlike most other morbid conditions when during the physical examination or history-taking a lead may be gained and followed up from general interrogation, this portentous state of body will remain unguessed until active work is commenced, unless the direct thought is in the clinician's mind and the question asked and developed, "Is the patient a bleeder?" In case of subsequent legal action by parents or relatives after an unfortunate ending to some simple cutting operation, it would be fair to suppose that a jury in relieving the attending surgeon of responsibility would perforce consider the shortcoming of failure to learn of the presence of the contraindication to the use of the knife along with the manifest peculiarity of the condition itself. Added weight in favor of the operator I believe would be that degree of necessity for the operation which had been undertaken. Death following one designed for plastic or mere cosmetic effect would doubtless be more liable to be adjudged worthy redress than those regularly required to relieve physical distress.

A case of hemorrhagic diathesis lately under my observation in which fortunately the outcome was satisfactory, though I was remiss in forgetting to ask concerning the possibility of the condition before beginning active work, concerned George P., an infant, aged twenty-one months, suffering from a constricted adherent foreskin. The child was brought for examination and treatment for general fretfulness. After a proper preparation and etherization I removed the foreskin and frenum. I added the latter procedure purposely as a precautionary measure, having in mind the prevention of subsequent constriction of the meatus urinarius, which in this case was

naturally quite small. In a number of instances in times past in young adult Hebrews I have been forced to do meatotomies, owing to the presence of gradual frenal contractions, lessening the size of the external urethral orifice subsequent to the rite they had undergone in infancy. The functions of the frenum being by its elasticity to replace the reflected foreskin and to prevent the last drops of urine after urination from entering the preputial sac by capillary attraction, I do not hesitate to sacrifice this structure in my performance of circumcision. The child stood the operation well and reacted promptly. Aside from the thought that came to me during the work that its blood seemed rather watery, the case presented nothing uncommon. At the second visit I developed the fact that the child was a "bleeder," the mother volunteering that she had forgotten to tell me that it ran in their family to bleed readily after the slightest scratch or cut. As the cut surfaces were in close approximation I saw no necessity for inserting more sutures, believing that their application would give rise to additional hemorrhage, but relied upon ice compress, tincture of iron chloride, hydrogen dioxide (though the wound remained free from purulent infection throughout), and tight bandaging. During the course of the next three weeks the condition of the little patient, and his readiness to bleed from the parts upon the slightest occasion, gave me some concern. Scabbing over of the meatus took place after every hemorrhage, and at one period prevented the child's urinating for some eighteen hours, but the consequences of softening the crust I knew would be so prompt that I preferred a stretched bladder to a bloodless child. Gradually union between the cut surfaces took place, with a delicate skinning over the frenal region. After the first week the hemorrhage came principally from about the site of the diminutive frenal artery. Under hemophilic conditions a lacerated frenal artery is as important a floodgate of the individual's body as an incised axillary artery would be under ordinary circumstances. In the case of the former to apply a ligature externally is to step into a quicksand, and it produces a curious but subduing effect to one's self-confidence to see one leak traded for three ooze-streams. The condition in the present instance matched a case which occurred under my care six or seven years ago, when I failed to secure properly a divided frenal artery in an adult whom I had circumcised. The individual re-

turned to me a few hours after the operation, shocked and bleeding. Loosening his blood-soaked clothing, fully a pint of jelly clots were removed and the vessel found freely jetting beneath my dressing. In the first instance I could not tie; in the last I did not. The former operation taught me carefully to consider this small but active vessel, and the last experience will I think enable me for a long time to come to recall the possibilities of hemophilia when considering operation.

The family history of the little patient was developed during the course of my care of him. The child's father and mother are living. The male parent has no history of bleeder's disease in his ancestry. The mother, aged 38 years, began to menstruate at sixteen, and has borne three children. The eldest is a male, now 16 years old, whom his mother describes as having been an "eight-months baby," and that soon after birth he underwent a "stretching" operation for phimosis. The second child is a girl of fourteen, who up to the present has had no sign of menstruation.

All of the children were breast-fed. The mother informed me that a breast-pump was necessary for her relief after each child was weaned, for a period of at least a month. Her "courses" continued throughout lactation in every instance. The older boy is delicate and bleeds freely, as does the mother, after the slightest wounding. The latter related that after her first confinement she bled so profusely that she was nearly drained of the vital fluid, necessitating that she be, as she expressed it, "packed in ice." The mother since early childhood has been troubled with frequent and profuse nosebleed arising from no apparent cause. The woman's physical appearance is good. While of slight figure, she does not impress the observer as being frail, neither is she anemic-looking. Relating back to the third generation: The woman's mother was of English stock and had reached the age of twenty-one before beginning to menstruate. Ignorant of the signs of the establishment of a natural function, the proverbial "washing of stained linen in a brook" took place, with a result that the girl spent three months in bed, suffering from an inflammatory attack.

From the marriage of the grandmother resulted seven children, three boys and four girls. One of the males is dead. A second is alive and a bachelor at forty-five. The third at fifty-four has a son

aged fifteen and a daughter sixteen. Three other children died in infancy. Of the living children both enjoy good health so far as known. Of the three sisters nothing is known.

The present history I should cite as one depicting mild diathetic defect. How far artificial feeding or the production of labor prematurely would affect the nutrition of an infant so as to enable it to have a better chance of survival to adult life it is impossible to say. It is notable in the present history, however, that the mother had a superabundant supply of milk.

Upon the whole, the outlook for " bleeders " is generally believed to be bad, individuals affected seldom reaching maturity. The pathology is given as being a condition depending upon a defective power of contractility of the walls of the blood-vessels. It would seem also that a failure of the coagulable properties of the blood exists.

SOME SURGICAL RESULTS WHICH MAY FOLLOW IMPROPER FEEDING

A POSTGRADUATE LECTURE DELIVERED AT THE HOSPITAL FOR SICK CHILDREN, LONDON

BY EDRED M. CORNER, M.A., M.B., C.B.(CANTAB.), B.SC.(LOND.), F.R.C.S.(ENGLAND)

Senior Assistant Surgeon to the Hospital for Sick Children, Great Ormond Street; Surgeon to Out-Patients, St. Thomas' Hospital, London

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Ladies and Gentlemen: I would remind you of the old fable in which the various organs of the body quarrelled with the stomach because it appeared to be idle whilst they worked. But it was discovered that without the stomach they soon became weak and useless. Digestion and absorption—that is, nutrition—are the most important functions which a child must perform. The fable teaches that all the other parts of the body are affected by and dependent on these functions of the alimentary tract represented by the stomach in Æsop's tale. So that good nutrition will affect all parts of the body for good; and bad nutrition will affect all parts for evil. Hence, although we pay scant attention to good nourishment and growth, we must pay very much attention to improper feeding on account of its effects being deleterious and universal throughout the body. Further, childhood, in virtue of its property of growth, is the most important epoch in life to consider, since bad feeding will lead to bad growth, the seeds of future disabilities being laid down. It is not practical to consider within this lecture all the possible results of improper feeding, so that only those which come under the notice of the surgeon will be mentioned, and not all these. Stress will not be laid upon the diseases which are well known to result from improper feeding, such as rickets and scurvy, but attention will be directed to surgical conditions which are not generally recognised as being so caused.

The results of improper feeding may be due to faults on the part of the child, as when suffering from some ailment; the food, as when

it is unsuitable; or, most often, both the food and the child are at fault. The practical lesson to be learned is that, although a child may be supplied with the best food, it may be wholly unable to digest it, and so suffer from the consequent ills. Many and many a time symptoms have been found to occur in breast-fed children. It should be remembered that mother's milk is not a panacea for all infantile ills.

At birth, the meconium in an infant's bowels is usually sterile. After birth organisms quickly obtain admission and multiply in the intestines. The babe is unaccustomed to the action of their chemical products, and should the rate of inception of the organisms be great, the baby must suffer from intestinal disturbances. These organisms may have a very profound effect on the child and its feeding. Suppose a baby to be fed with its natural food (its mother's milk), it will be able to digest and utilise a certain amount. Should there be any excess of this amount, the organisms in the bowel will cause its decomposition, just as they would do if outside the body. For our purpose, the fermentation of proteids, carbohydrates, and fats in the intestine may be compared with the fermentation of sugar by yeast. When sugar undergoes fermentation, two substances are formed, amongst others, alcohol and carbonic acid gas; that is to say, a toxin and a gas. When proteids, carbohydrates, and fats ferment, in a similar way, toxins and gases are found amongst the many other products. Any residuum of nourishment, even if it is of the very best, will go bad and ferment. If it is quickly defecated, all is well; but if it is retained and the condition remains for some time, then the child's health will suffer. Hence, it is the overfed rather than the underfed baby which develops rickets. The children of the well-to-do classes are more abundantly fed than are the children of the poor. Hence the great prevalence of slighter degrees of rickets and other ailments amongst the former. Similarly, rickety babies are frequently fat and not lean, the result of the plenteous supply of food.

Seeing that proper as well as improper food can become poisonous when it decomposes, too much care cannot be given to the condition of the bowels and the character of the motions of a young child, as they will indicate the conditions present.

There are three lines to be followed in considering the conse-

quences of food-fermentation within the bowel; namely, the effects of the toxins, the effects of the gases, and, lastly, the effect of the fermentation on the bowel itself.

The Effect of the Toxins.—The poisonous products, the toxins, are absorbed from the intestine and carried by the portal vein to the liver, which is the first organ, apart from the alimentary tract, to be affected. As you know, the liver usually becomes enlarged. But its enlargement takes time, and the general system during this period is being poisoned indirectly through the hepatic veins, and directly through the intestinal lacteals, the receptaculum chyli, and the thoracic duct. It is obvious that this is the stage when the child's condition may be most advantageously diagnosed; treatment is then easier and more sure in its effect. This stage can be recognised, one being more likely to be misled by its frequency than by anything else. The symptoms are: The child is querulous, often crying and not taking its food so well, it does not sleep well, its pillow is often wet from perspiring about the head, its bowels are inactive or loose, its abdomen is large and may show multiple hernias, divaricated recti, and bulging flanks; it suffers from wind, and the stools are offensive. To put it generally, the child is obviously uncomfortable and uneven in its methods of life and the performance of its various functions. Sickness indicates gastric irritation and, therefore, improper food, rather than excess of good and proper food. Hence it need not be present at all. And in spite of its improper feeding the child is often fat and apparently well cared for.

At this stage the treatment is easy. The diet must be attended to with regard to both quantity and quality; the bowels must be regularly evacuated, and the child's stools and general condition watched to see the results. As the fermentations are acid, it is good practice to combine the purgative with an alkali, as in a powder of rhubarb and bicarbonate of sodium. If it is recollected that when a child swallows a foreign body several days may elapse before it is passed, it will be realised what a quantity of decomposition can take place in food residues before they are got rid of.

Too much stress cannot be laid on the importance of the recognition and treatment of this initial phase of intestinal intoxication, or, as it might be termed if its ultimate results are borne in mind, the pre-osseous phase of rickets.

If this phase is allowed to pass, then the toxins will exert their actions on all the tissues in the body. When the bones are affected, the condition is recognised by the enlarged epiphyses, etc., characteristic of rickets. When the vascular system is affected, scurvy or anemia appears. Should both systems be affected simultaneously, the disease is called scurvy-rickets. But these diseases only represent the affection of two systems! We do not know what may be happening in the other parts of the body. If we were to guess from what we know occurs in the bony and vascular systems, we must infer that gross and grave changes are going on elsewhere. It is dangerous to hazard a guess as to what diseases or deficiencies of organs are thus brought about. But it is salutary to think that as great harm may be done in the brain and elsewhere in the nervous system as in the bones.

The Effects of the Gases produced by Fermentation.—As the meconium in the bowels of an unborn infant is usually sterile, no decomposition should have occurred and no gas should be present. When organisms obtain admission to the alimentary canal, fermentation will begin and gas will be produced. A certain amount of fermentation is essential and seems physiologic to the adult organism. One effect of the gas formation is to increase the intra-abdominal tension, the effect of which will be most felt on the weaker parts of the abdominal wall. These weak parts are the linea alba, the umbilicus, and the inguinal regions. If the gas formation is large, hernial protrusions may appear in all these regions. With regard to the inguinal region, it matters not if a vaginal or funicular sac is already present, it is the increased intra-abdominal tension which forces the bowel or omentum into the sac. Otherwise, the tension has to accomplish the additional task of forcing a peritoneal sac down with the hernia; this would be an acquired hernial sac. It is not unusual that when a congenital abnormality occurs it is accompanied by other abnormalities. But when two inguinal hernias, an umbilical, and a protrusion between the divaricated recti are present, it is more logical to associate their presence with one common cause rather than many. Especially is this so when there is also a large abdomen and bulging flanks. There can be no doubt that many of the hernias of children arise in this way. But as it is impossible to distinguish between the funicular and the acquired sac, it is impossible to say how many. Nevertheless, it is a fact that

simple treatment will cure many of these hernias and relieve a number more, obviating the immediate need for operation.

If these hernias or the abdominal distension are present for a long time, they, or it, will lead to permanent weaknesses on account of the fact that they have modified the normal healthy growth which should take place in the structures of the abdominal wall. Hence, if they are allowed to persist they may sow the seeds for the development of a hernia in the adult. The relief of the distension and the cure of the hernia whilst the child is still young are very important to the welfare of the adult.

There is still another important effect besides that on the hernial rings, which gas formation and prolonged distension will produce, namely, the effect on the abdominal wall. The back of the abdomen is well supported by the spine and its muscles; the lateral part of the abdomen is also well supported by the ribs, ilia, and muscles; the front of the abdomen is supported only by long muscles, and in consequence bears the brunt of the abdominal distension. The recti are long strips of muscle with bony attachments at either end; and, as such, suffer comparatively little. The portions of the external and internal obliques, particularly the latter, which come into consideration here, have only one bony attachment, and, being flat, broad muscles, are stretched by the abdominal distension both lengthwise and broadwise, causing some separation of their component muscular bundles. Now, if you stretch an elastic sheet in both its superficial directions, when the stretching forces are removed the sheet returns approximately to its former size. But if instead of a passive elastic sheet, an actively growing muscle is stretched for some time, on account of the multiplication and growth of its fibers that muscle will not return to its original length when the stretching forces are removed, having by its growth adapted itself to the increased length. Hence, after a child's abdomen has been distended for some time, the external and internal obliques, the latter muscle suffering the more, are permanently stretched and redundant. This is evidenced by the bulging flanks seen in these cases. By this means another source of weakness may be transmitted from the child to the adult, rendering the latter disposed to hernia formation, should such not already have occurred.

The Effects of Fermentation on the Intestine.—The decompo-

sition of food residues in the intestine leads to numerous changes in it. Gastritis indicates that improper food, rather than excess of good food, is responsible for this condition. Jaundice indicates irritation of the duodenum. Enteritis and colitis show themselves by pain, diarrhea, mucus and perhaps blood in the stools. A secondary infection with tubercle baciili may lead to tabes mesenterica. Intussusception and prolapsus recti may occur. As the child has abundance of lymphoid tissue about its cecum and appendix it usually escapes appendicitis, such as would occur in an adult. The above are all changes within the bowel, and are accompanied by some subacute inflammation of the coats of the bowel. The inflammation of the mucous coats shows itself in diarrhea, mucus and blood in the stools. Inflammation of the muscular coat leads to inaction of the bowel and constipation. Inflammation of the peritoneum over the bowel leads to the presence of a small quantity of fluid in the peritoneal cavity; and, should a patent processus vaginalis be present, to the formation of a hydrocele of the tunica vaginalis—the common hydrocele of children.[1]

The Origin of Some Cases of Varicocele.—We have now finished our brief sketch of the surgical results of improper feeding. There are one or two problems still to consider. The first is that of varicocele. It is not customary to look for the origin of varicocele in improper feeding. It is a surprising fact that right-sided inguinal hernias form about 60 per cent. of all the hernias of children. The presence of a hernia in the inguinal canal, unless it is tightly prolapsed and nearly strangulated, will remove, on account of the dilatation, any obstruction to the return of the blood in the veins of the right pampiniform plexus. Therefore, there is reason that the right side should not be affected with varicocele. On the other hand, we know that the left inguinal region does not give way, leading to the formation of the hernia so frequently. So it will be of interest to see what will be the effect of this not giving way. The inguinal canal has a valve-like action, as seen in Figure 1. If a hernia forms the valve is destroyed; if a hernia does not form when the abdomen is distended the valve is not destroyed, but is closed tighter than before, obstructing the venous flow in the pampiniform plexus. We

[1] International Clinics, 1905, ii, p. 155, Fig. I. C.

see, therefore, that there is reason to expect the right side to be free from varicocele; whilst there is reason to expect it on the left. If care is taken at operations on children to observe the veins, especially those of the left pampiniform plexus, it is not uncommon to find miniature varicoceles in quite young children. Such observations support the above view that the seeds of varicocele are often sown in childhood.

Another great cause for the predominance of left-sided varicoceles is that the left pampiniform plexus has two venous supplies—namely, from the left spermatic vein, which joins the left renal

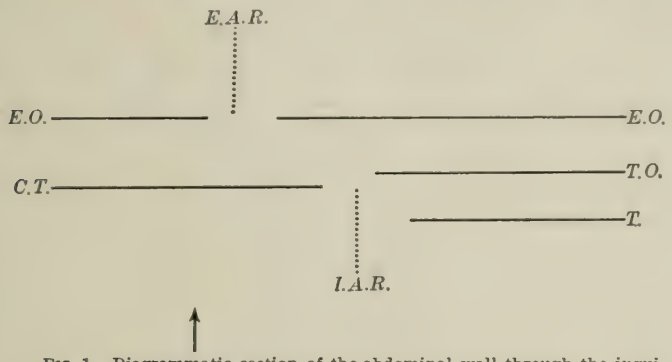

FIG. 1.—Diagrammatic section of the abdominal wall through the inguinal canal, showing its valvular relation to intra-abdominal tension. *E. O.*, external oblique muscle; *T. O.*, the internal oblique; *T.*, the transversalis; *C. T.*, the conjoined tendon; *E. A. R.*, the external abdominal ring; *I. A. R.*, the internal abdominal ring. The arrow indicates the direction of pressure which will close the canal.

vein, and a vein (or venæ comites) which accompanies the spermatic artery and opens into the inferior vena cava.

The varicose veins of the pampiniform plexus of the child remain unnoticed clinically until puberty, when the testis takes on rapid growth and functional activity. The condition of the veins, on account of the increased work which they have to do, becomes patent, and is called a varicocele. There can be little doubt that the presence of varicoceles in young adults can often be explained on this hypothesis. Though it would be foolish to attempt to explain all varicoceles in this way, there is a further stage in the development of a varicocele to which attention may be turned. If the venous obstruction is chronic and attains sufficient degree, it will set up a chronic inflammatory condition of the tunica vaginalis leading to the

formation of a hydrocele. In this way, the occurrence of hydroceles in young adults receives explanation; as does also the fact that they recur and recur after simple tapping. The production of hydroceles, spermatoceles, and other changes as the result of venous obstruction is well illustrated in the paper by Mr. Nitch and myself.[2]

From the above sections it will be seen that the seeds for the formation of hernias, varicoceles, and hydroceles in adults can be sown in childhood, another example of the fact that the child is parent to the adult.

The Nasopharynx and Adenoids.—If one thinks over the rickety affections of the skull which are known, it becomes obvious that we recognise gross changes as occurring in the vault of the cranium, whilst no attempt has been made to ascertain if any changes are to be found in the base of the skull. Of course, the vault is developed in membrane and the base is laid down in cartilage. But we know that bones developed in cartilage suffer from rickets, as can be seen in other parts of the skeleton. So there is every reason to expect that bony deformation will be present in the base of the skull. The base of the skull is an awkward situation to study on account of the difficulty of making observations. The bones, as elsewhere, must become thickened and, later, sclerosed. What will be the effect of this? The nasopharynx will become encroached upon and rendered smaller, making it difficult, or at least uncomfortable, to breathe through the nose. If the obstruction is sufficient to cause a negative pressure in the nose, the surrounding softened membrane bones, such as the nasal and palate bones, will fall in. The nose will sink and the palate become high, surely converting the child into a mouth-breather. The unventilated nasopharynx forms a kind of cul-de-sac, becoming the breeding-ground of the organisms which form the flora of the nose and mouth, such as the appendix does for the large intestine. The irritation afforded in this way leads to the growth usually called the overgrowth of the lymphoid tissue present, giving rise to the condition we recognise as adenoids. The nasopharynx is now stuffed up with adenoids and the bones have enlarged and fallen in, stopping nasal respiration. If a limb is functionless, it will grow imperfectly; so with the nasopharynx. That cavity, having been rendered functionless, grows much less than it would if it had

[2] Lancet, Jan. 27, 1906, pp. 191–193.

been functional. The thinness of the nose, the poor development of the upper jaws, etc., seen in adults with neglected adenoids is usually attributed to this. But there is another point to which I would direct attention, namely, the influence of rickety changes in the bones and the absence of nasal respiration upon the development of the air cavities in connection with the nose, such as the sphenoidal sinus. The imperfect development of these air-containing spaces will aid materially in stamping upon the face of the adult the stigma of neglected adenoids, the hallmark of imperfect growth and function.

From the above, it will be seen that there is a very intimate connection between improper feeding, rickets, and adenoids. Further, the relationship illustrates that the proper performance of its functions alone leads to the proper growth of the part, whilst bad physiology in childhood leads to bad development—*i. e.*, anatomy; a lesson which should always be kept in mind when treating deformities of body, function, or mind in children.

If one keeps in mind the stage of intestinal toxemia before rickety changes have already declared themselves in the bones, one will realise how often cases sent as adenoids merely want dieting and treatment, to be followed by a course of nasal respiratory exercises, and do not require operation. Knowing that obviously rickety bones become perfectly straight as the result of treatment and healthy growth, the proper treatment must be adopted and time allowed to elapse for the rickety changes in the bones of the nasopharynx to be removed, before a final prognosis can be given. Certain is it that many of these children avoid the need of all operative treatment, whilst if the medical treatment is not adopted they come to surgery for aid, but alas, owing to the previous neglect, it is often impossible to do a great deal of good.

The Relation of Congenital Syphilis to Rickets.—There can be no doubt that there is a very intimate relationship between these two diseases. An authority writes that "congenital syphilis is so often a disease in which general malnutrition is the most prominent feature." There is no statement more truthful than this, and in it lies the kernel of the confusion of these two diseases. It is the "malnutrition" which produces the rickets. The syphilitic child may not be able to digest and absorb the very best of foods which can

be given to it. Further, if the accounts of the two diseases are read side by side, it will be seen that there is much in common. For instance, the skulls are similarly malformed, they develop similar nodes, the long bones are similarly affected. In both diseases the vault of the skull shows the condition of craniotabes. The characteristic of irregular ossification in a bone developing in cartilage will not be very noticeable, but should the bone be developed in membrane, owing to the irregularity, it must present hard and soft areas, such as are described under the name craniotabes. It is now obvious why that condition has been noticed in the thinner portions of the cranial vault which are developed in membrane.

The same authority quoted above says also: "It will be noted that considerable resemblance exists between the bony lesions due to syphilis and those due to rickets; the latter, however, occur usually at a later period." The two diseases, certainly, are confused. In fact, it can be stated that every syphilitic child must suffer in some degree from "improper feeding," frequently to the extent of developing rickets; but it is only a very small percentage of rickety children who are syphilitic. To diagnose infantile syphilis it is necessary to recognise definite points and stigmata which cannot be attributed to rickets. There can be no doubt that congenital syphilis is a maligned disease, having much attributed to its specific action which is due to the rickety conditions which it may produce.

Improper feeding in the adult produces its results just as it does in the child. The absorbed toxins cause changes in the liver,—*e. g.*, cirrhosis, and general fatness, and obesity. The gas-formation causes the distended, protuberant abdomen. So that the fat, pot-bellied adult is the mature type of the deformed, rickety child.

Summary.—There is no more important function for its welfare performed by a child than that of nutrition. The foregoing lecture has brought forward so many practical points that it has been thought best to epitomise them.

(1) The best foods unwisely administered can produce symptoms of improper feeding. They can often be seen in breast-fed babies.

(2) The fault may be with the child, and not with the food.

(3) At birth the alimentary canal is sterile. The admission of

organisms begins the decomposition of food residues and the intestinal education of the child.

(4) There is a recognisable stage of toxemia before any rickety signs are manifest. Treatment should be adopted then.

(5) The toxins are known to produce gross changes in some organs; probably they do so also in other structures as yet unknown to us.

(6) The gas formation may produce inguinal or umbilical hernias, divarication of recti, a large abdomen, bulging flanks, hydroceles, etc.; sowing seeds of weakness for the possible future development of hernias, varicoceles, hydroceles, spermatoceles, etc.

(7) In the intestine itself many changes may occur—gastritis, enteritis, appendicitis, colitis, intussusception, prolapse, etc.

(8) Adenoids are commonly a sign of rickets or improper feeding.

(9) Congenital syphilis is held responsible for many rickety changes.

DISORDERS OF THE UMBILICUS, WITH SPECIAL REFERENCE TO THE NEW-BORN AND THE INFANT: II. UMBILICAL FISTULAS, SINUSES, AND CYSTS*

BY A. ERNEST GALLANT, M.D.

Professor of Gynecology in the New York School of Clinical Medicine; Consulting Surgeon to the Jamaica Hospital, New York; Assistant Gynecologist to the Roosevelt Hospital Out-patient Department, New York; Formerly Special Assistant in the Department of Surgery (Professor W. W. Van Arsdale) of the Good Samaritan Dispensary, New York City.

VESICO-UMBILICAL, URINARY, URACHRAL FISTULAS

Umbilical fistulas are distinguished by the character of the discharge, and may be grouped as:

(1) Fecal: (*a*) diverticular; (*b*) inclusion—intestine.

(2) Gastric: inclusion stomach.

(3) Biliary: umbilical vein or gall-bladder.

(4) Hermorrhagic: umbilical arteries.

tion.)

(5) Urinary—urachal.

FECAL UMBILICAL FISTULAS

When the omphalomesenteric duct fails to atrophy and remains patent through its length, communicating with the intestine (Meckel's diverticulum), or when the intestine protrudes through the umbilical ring into the cord, and is included in the ligature, then, after the cord falls, a discharge of feces and intestinal gas will occur through the navel which will be the seat of a bright red, nipple-like growth (Fig. 9, B). As time goes on further eversion may take place and finally involve the whole caliber of the gut, as shown in Fig. 9, C, D. In the earlier stages there is apparently but one opening, but a probe may be passed up and down; and in stage "D" all the contents of the bowel will be evacuated through one arm of the tumor.

* I am much indebted to Dr. Eleanor Tomes and Dr. H. T. Blumensohn for most of the references to original articles in French and German.

An umbilical adenoma, whether arising from Meckel's diverticulum, or intestinal inclusion according to Thoma, "has all four of the intestinal coats, mucous, submucous, muscular, and serous."

Fecal fistula at the umbilicus, due to intestinal occlusion below, has been reported by Clayton.[22] All the feces passed through the umbilicus; the rectum and anus were normal; digestion imperfect. The child died on the twenty-second day. The autopsy showed imperfect development of the intestines below the diverticulum leading from the fistula, while the portion above was normal.

"The so-called Meckel's diverticulum is always found in the form of an intestinal appendage, attached somewhat obliquely to

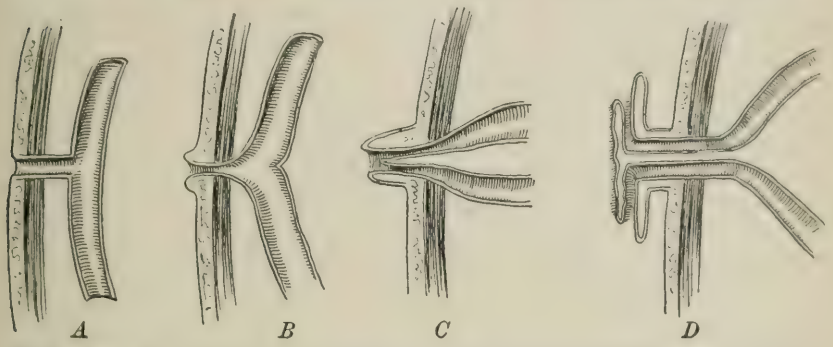

FIG. 9.—Stages in the prolapse of the intestines through an umbilical vitelline fistula. (Redrawn after Barth.)

the lowest portion of the ileum, from 0.3 to 1.3 cm. above the ileocecal valve. It is from 2 to 15 cm. in length, and from 1 to 5 cm. in diameter. Sometimes, however, it has a greater diameter than the small intestine. It is usually of a cylindrical, a conical, or a knoblike form. At the junction of the diverticulum with the ileum there is often found a valve which Meckel interprets as an attempt at closure of the intestine. The diverticulum sometimes ends in a ligamentous cord or in several threads, which are either free in the peritoneal cavity, or adherent to the inner surface of the umbilicus or to some other part of the peritoneum. In some cases the ligamentous cords and threads, or a pervious canal, do not pass from the inner surface of the umbilicus to the apex of the diverticulum, but directly to the mesentery. The

[22] Birmingham Med. Rev., Eng., April, 1895.

true (Meckel's) diverticulum without connection with the abdominal walls occurs regularly in the coot, the snipe, and the swan" (Cazin [23]).

Treatment of Fecal Umbilical Fistula.—Owing to malnutrition which is usually associated with this condition, if permitted to continue, the radical operation carried out by Stierlin [24] is "the only treatment" which promises the best result: Male child, aged 2 months. After the cord separated the mother noticed a red tumor at the navel, out of which came fecal matter. At the time of operation the child was well developed. A tumor the size of a hazlenut projected, penis-like, at its base 4 to 5 mm. in diameter, the color of intestinal mucous membrane, surrounded by a thickened ring of skin. The navel lay a little to the right side. Pressure upon the abdomen caused the escape of gas and feces. The sound passed directly in 6 to 8 cm., a rubber catheter, 25 cm., showing communication with the small intestine. At operation a cylinder 11 mm. wide and 9 cm. long attached to the small gut, with omphalomesenteric artery running up to the umbilicus, was found; the diverticulum was removed, and the end sewed as in any intestinal resection. The patient was discharged in ten days, cured.

W. H. Battle [25] was not so fortunate. A female infant, 1½ years of age, presented a pear-shaped tumor at the umbilicus, which was present since birth; length 1½ inches; red, smooth, covered with mucous membrane; bleeds on manipulation. Probe passes into opening 2 inches. Slight, thin discharge, of rather feculent odor; some eczema of the skin surrounding the aperture. Mass irreducible. Bowels move normally. Operation: Chloroform; 1½-inch incision. Intestine and diverticulum drawn out; the latter cut off ¼ inch from intestine; the stump invaginated and closed by the Lembert suture. Next day the child developed scarlet fever and died 9 days later. Post mortem showed abdominal wound healed; peritoneum healthy; no adhesions.

George Heaton [26] operated in the same way, on a similar case

[23] Tillmanns' Regional Surgery, Tilton, p. 9.
[24] Deutsche med. Woch., 1897, xxiii, 188.
[25] Trans. Clin. Soc. Lond., 1892–3, xxvi, 237.
[26] Brit. Med. J., 1895, i, 473.

and at autopsy found both large and small intestine below the diverticulum much contracted.

Early operation, before contraction takes place, will give the best result.

Shepherd [27] reports a fecal fistula from a diverticulum, which projected about an inch from the umbilicus of a male child, through which there escaped liquid feces. By abdominal section the root of the diverticulum was removed, and the opening in the bowel closed by a double row of Lembert sutures; and the child recovered.

Gambert [28] reports an anteroumbilical fistula, which was noticed on the fifth day, as a granulating tumor, through which gas and feces passed, and which he attributed to ligature of the cord; the omphalomesenteric canal extended into the cord. Gambert applied the actual cautery to the granulating surface, and the fistula closed later.

VITTELINE MUCOUS SINUSES

When the omphalomesenteric duct fails to become obliterated at its umbilical end it will make itself manifest by a mucous discharge through the umbilicus, as in Broca's case [29] which presented a sinus 4 cm. deep, discharging mucous, but no feces; the intestinal end was not open, but was connected by a cord. Broca excised the sac, cut away the cord-like remnant, and closed the stump by lateral enterorrhaphy. Recovery ensued. (Fig. 10, F.)

These sinuses may present as adenomatous projections at the umbilicus, due to prolapse of the omphalomesenteric remnant, as in a case recorded by S. W. Wheaton [30] in a male child 10 weeks old. The tumor was ½ inch wide, 1 inch long, and attached by a narrow pedicle to the umbilicus. There was a constant mucoid flow from the orifice, through which a probe passed downward and backward for 2½ inches. This was removed by ligature, and silver nitrite was applied to the stump. Four months later the child was quite well, the umbilicus healthy and no cyst could be felt.

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[27] Montreal Med. Jour., 1891–2, xx, 539.

[28] Archives de Tocologie et de Gynaecologie, Paris, Dec., 1893.

[29] Rev. de Sciences Med., Paris, April 15, 1895; Sajous Annual, 1896, ii, 1–25.

[30] Trans. Obstet. Soc. Lond., 1893, xxxiv, 184.

Railton [31] reports an instance of prolapse of the omphalomesenteric remnant in a child of 11 months. For months there had been

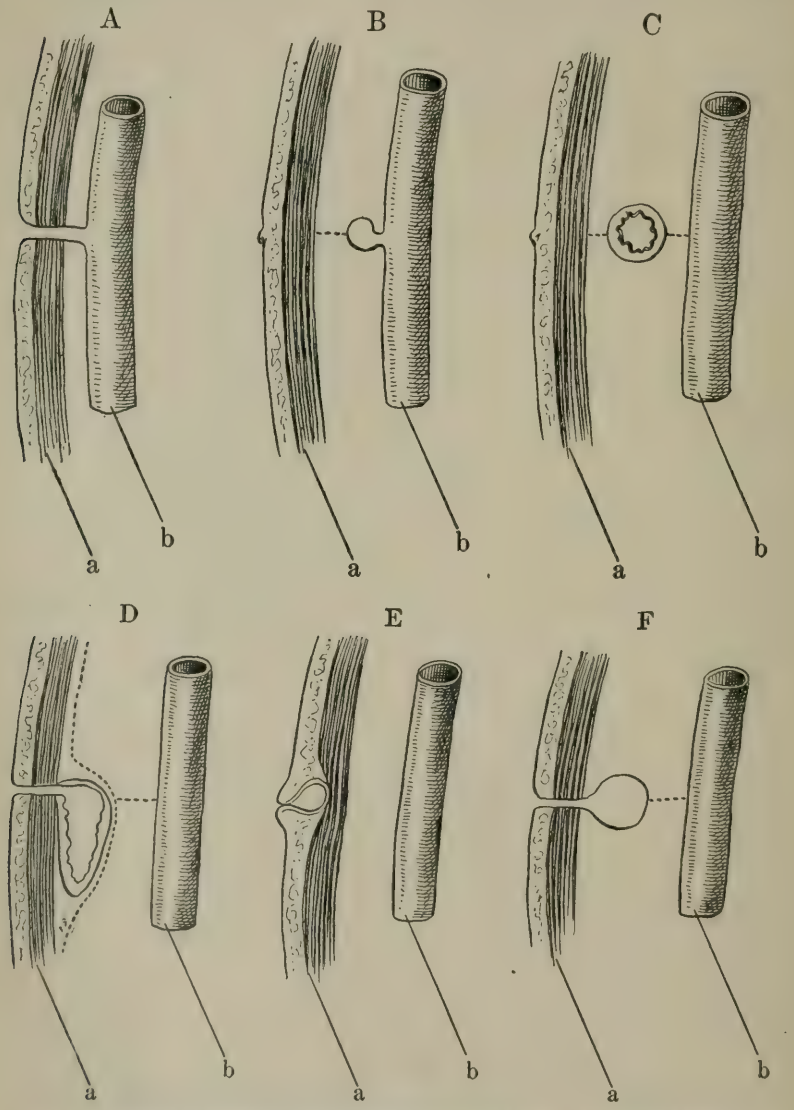

FIG. 10.—Diagram of different forms of persistent vitelline duct. A—Vitelline fistula: B—Meckel's diverticulum; C—Roth's mucous cyst; D—Roser's preperitoneal cyst; E—Zumwinkel's subcutaneous cyst; F—Broca's mucous sinus. a—Abdominal wall; b—Intestine.

[31] Brit. Med. Jour., 1893, i, 795.

a foul discharge through the umbilicus, which latterly had disappeared, leaving no aperture. At the time he saw it there was only slight moisture and the child seemed perfectly well. A small, smooth projection, ½ x ⅛ inch, thick, red, cylindrical, and covered with mucous membrane. This was ligated at its base, snipped off, and three days later all traces had disappeared. The outer layer was mucous membrane and contained glands of Lieberkuhn. (Fig. 9 B.)

Under the caption of " Subcutane Dottergangscyste des Nabels " Zumwinckel [32] relates having seen, in a girl of 7 years, a small

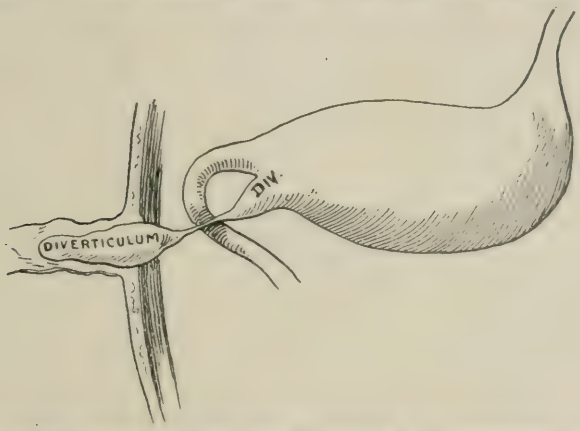

FIG. 11—Diagram representing the origin of prolapse of the mucous membrane of the stomach through the umbilical ring. (Redrawn after Tillmans.)

cyst beneath the skin and lying on the fascia, which proved to be a blind pocket leading from beside the navel. The cyst-wall showed the same structures as the small intestines. (Fig, 10 E.)

GASTRIC UMBILICAL FISTULA

Instances of this rare and interesting congenital prolapse of the mucous membrane of the stomach through the umbilicus have been recorded by Tillmans, Roser, Liegenbeck, and Wölfler as met with, strange to say, in boys at 13, 1½, 2½, and 7 years respectively. Tillmans first saw the boy when at 13 years of age. It was first noticed 4 to 6 days after birth, when the cord fell, as a red tumor, about the size of a cherry. At the time of operation it presented as a pedunculated tumor the size of a large walnut, covered with

[32] Arch. f. Klin. Chir. Berlin, 1890, xl, 838.

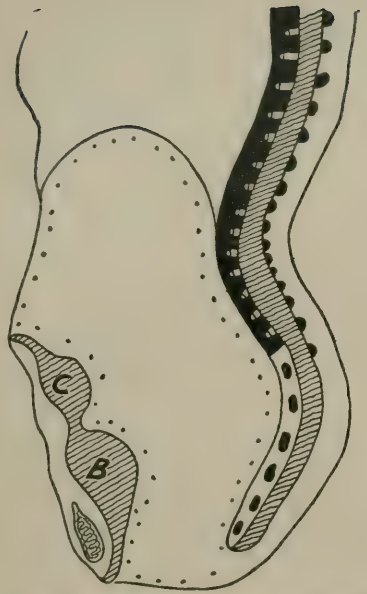

FIG. 12.—Urachal cyst of a 10-year-old boy from post-mortem. (Robinson.)

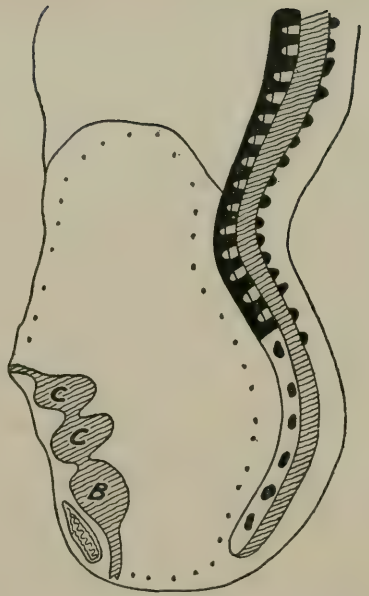

FIG. 13.—Multiple urachal cyst. (Robinson.)

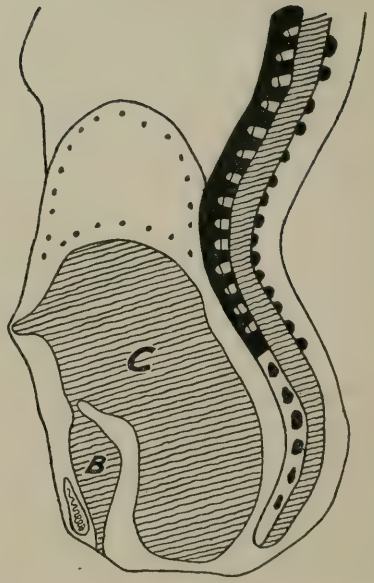

FIG. 14.—Urachal cyst which dips into the pelvis. (Robinson.)

The dotted line is to represent the peritoneum. *B*, bladder; *C*, urachal cyst.

(Annals of Surgery, 1891, vol. xiv., p. 339-349-340.)

mucous membrane, discharging an abundant quantity of secretion, which on microscopic and digestive experimentation proved to be a stomach diverticulum (Fig. 11) which had been tied off with the cord, and remained open at the umbilicus. Operation disclosed the fact that the gastric end had atrophied to a mere thread, occluding the stomach contents; this was severed, the stump cauterized, and the whole sac excised, resulting in a complete recovery.

"In mammalia, the visceral laminæ close the abdominal cavity, the allantois is thereby divided at the umbilicus into two portions: the outer part, extending from the umbilicus to the chorion (Figs. 12, 13, and 14) soon shriveling; while the intra-abdominal portion goes to make up the urinary bladder and a remnant, extending between the fundus vesicæ and the umbilicus—the urachus. After birth the umbilical cord and with it the external shriveled portion of the allantois, are cast off at the navel, while the urachus atrophies and normally remains as an impervious cord stretched from the top of the bladder to the umbilicus, in the middle line of the body, immediately beneath (external to) the parietal layer of the peritoneum. It is sometimes enumerated among the ligaments of the bladder." (Foster.)

Luska [33] states "that in the majority of males the urachus is found to be partially open and lined with mucous membrane"; changes in its calibre may take place at any age, . . any part of the urachus may dilate, but the part next the bladder seems the most liable, as that is the last portion to become functionless.[34]

Congenital Vesico-Umbilical Fistula: *Absence of the Sexual Organs.*—Obliteration of the urachus should occur about the fifth month of fetal life, but that this does not invariably take place is shown by a case of Preston's [35] which he saw a few days after birth, in an otherwise healthy, well-developed child, with a complete absence of the sexual organs.

The shape of the orifice in urachal fistulas as detailed by Henry Morris [36] in a "Clinical Lecture on the Surgical Affections of the

[33] Virchow's Arch. 1862, xxiii, 1.
[34] F. B. Robinson, Annals of Surg., 1891, xiv, 337–355.
[35] New York Med. Record, 1898, liv, 315.
[36] Clinical Journal, Lond., 1892–3, i, 165.

Umbilicus" varies very much: (*a*) Button-like, papillary, or columnar projection; (*b*) the urine may escape at several points on the surface of the hernial protrusion of the tube; (*c*) a hernial protrusion may act like a plug, having a single fistulous opening at the side of its pedicle (Petit's case); the urine accumulates, escapes when voluntary effort is made, jets out forcibly through the umbilical opening though it does not continue to escape thereat throughout the whole act; (*d*) the orifice may be a single, circular, oval, or angular deficiency of the integument and other tissues; (*e*) a cup-like depression hidden by the falling together of the folds of the umbilicus; (*f*) the tract of the fistula may be very circuitous, and the orifice a simple slit or puncture.

Closure of urachal fistulas may take place spontaneously; but it may also lead to serious consequences, as in Hoffmann's case,[37] a man, seen when 28 years old. A urinary fistula had been closed by caustics when the child was three years old. During the past year a tumor had developed between the symphysis and umbilicus, the size of a goose-egg, and finally perforated into the peritoneal cavity, causing a peritonitis which resulted in his death.

Treatment of Vesico-Umbilical Fistula.—In 1826, Roux[38] saw a boy of two years suffering from a congenital fungous tumor of the umbilicus, of a livid red color, and an orifice in its center, through which some urine escaped; but most urine was passed through the penis. He passed a sound into the urethra, and covered the umbilicus with an elastic ball, but it caused ulcerations, and the child left the hospital without further treatment. Starr,[39] Meyer-Kempen,[40] W. J. Penny[41] proposed operation in similar cases but were not permitted to do so. Paget,[42] on June 29, 1858, in a female child, with incessant flow of urine through the umbilicus, freshened the edges of the orifice, inserted a pin and thread in figure-of-eight, and when the pin was removed three days later the urine still flowed. The condition is said to have been cured in two years(?).

[37] Arch. für Heilk., 1870, xi, 373.
[38] Arch. gén. de méd., 1826, xi, 554, obs. xix.
[39] Prov. Med. Journ., Lond., 1843–4, vii, 288.
[40] Casper's Wochenschr. fur die ges. Heilk., 1844, 424.
[41] Bristol Med.-Chirg. Journ., 1888, vi, 36.
[42] Medico-Chir. trans., 1861, xliv, 13.

That in case there be no obstruction to the outflow through the natural channel, a fistula may close is illustrated by Smith's case,[43] a boy of two years, with urine passing all the time through an umbilical fistula and was cured by simple ligature.

Gueniot,[44] saw a male child, 10½ months old, micturition normal, but urine passed through the navel when child cried. Compression and astringents were ineffectual. A thread was passed through and the stump cauterized repeatedly, and silver nitrate applied every 3 or 4 days, for ten days, with complete recovery in five weeks. On the other hand, in Gueniot's second case, a female 18 to 20 years, there was no opening into the bladder, all urine passing by the umbilicus, and it was found necessary to remove a membrane which closed the urethra and ligature the umbilicus. The girl recovered in 12 days.

Urachal fistula due to phimosis causing overdistention of the bladder and cured by the circumcision have been recorded by Stadfeldt, I. I. Charles, Lexer. In the latter's case after circumcision the fistula closed spontaneously.

Urachal Fistula; Imperforate Urethra.—J. L. Petit[45] cites an instance of a boy without a urethra, whose urine discharged involuntarily through the penis and umbilicus at the same time. Petit also tells of a female, born with a "closed urethra" and a tumor the size of a cherry at the navel, by lifting which an opening can be seen through which the urine escaped. Shattuck[46] describes a male fetus of about the fourth month, with "imperforate urethra." The abdomen was greatly distended with fluid; complete atresia of the bladder; both kidneys cystic; general cystic disease.

Urachal Fistula; Bladder Calculus.—Early operation is important in cases such as that of Page.[47] The urachus remained open and a ring-shaped calculus had formed upon a hair in the bladder. Urine had escaped through the umbilicus since birth. When the man was forty years old, 15 years after removal of the stone, Paget freshened the edges and closed the fistula: Forty years of discomfort, which might have been avoided by early operation.

[43] Med. Times and Gaz., 1863, i, 320.
[44] Bull de Therap., 1872, lxxxiii, 299.
[45] Oeuvres Completes, Limoges, 1837, 799.
[46] Trans. Path. Soc. Lond., 1888, xxxix, 185.
[47] Medico-Chir. Trans. Lond., 1858, xxxiii, 293, xlvj, 13.

Urachal abscess and fistula due to polypoid excrescence of the bladder was found in a male child which came under the care of E. Stanley.[48] The child suffered for several months with irritation of the bladder, which was thought to be due to stone. The whole of the urine, for a time passed through the umbilicus, none through the urethra. Post-mortem a number of soft, membranous, pedunculated growths were found projecting from the mucous membrane of the bladder and in great part filling it. Savoy[49] met with a similar case of abscess in a boy of 13 months, which later discharged pus and urine. Death occurred 9 days after opening of the abscess; and autopsy showed obstruction of the bladder. Weller Van Hook[50] cured an urachal abscess in a girl of 6 months, by dilatation of the umbilical opening, and tube drainage.

Littre[51] describes a girl of 12 years, in whom the urethra was closed by a membrane,[52] this being the first of its kind on record.

Among other conditions which lead to urachal fistula may be mentioned (*a*) Stone in the urethra in a boy of 18 years; and a man of 30, who had always passed urine by the umbilicus, supposed to be due to some obstacle in the urethra; (*b*) Enlarged prostate, in a man of 79 years;[53] (*c*) Gonorrheal stricture, with habitual retention.[54] Chapin[55] made his patient, a man of 66 years, in great agony from retention, happy by inserting a catheter through the urachus into the bladder which could be emptied at will by a stopcock. Leveque[56] reports an unusual case in a man of 80 years who for 25 years had discharged urine freely through both the umbilicus and urethra.

Chronic Cystitis and Fistula.—A girl of 8 years had suffered from dysuria since childhood; four months later she died from chronic cystitis, with hypertrophy of the bladder, to which cause Cadell[57] after autopsy attributes the urachal patency.

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[48] Trans. Path. Soc., 1852, iii, 127.
[49] Med. Times and Gaz., 1852, 33, 106.
[50] Am. Journ. Obstet., 1894, xxix, 624.
[51] Mem. de l'Acad. de Sciences, 1701, 23.
[52] See Sabatier's Traite d'anat., t. ii, 402.
[53] Levie, Jahresber. fur ges. Med., 1878, ii, 413.
[54] Holscher, J. P. Quoted by Douglas, loc. cit., 474.
[55] N. Amer. J. Homeop., N. Y., 1897, 3s., xii, 288.
[56] Journ. de med et de Chir., 1811, xxi, 124.
[57] Edinb. Med. Journ., 1878, xxiv, 221.

Urachal Fistula, Septate Bladder with Polyps.—Ball[58] tells of a boy of 10 years who suffered from incontinence of urine and hematuria. After failure to cure by cauterization and later ligature he transplanted a flap of healthy abdominal surface over the orifice at the navel. A few days later peritonitis set in which caused the death of the patient. The bladder contained a septum which was attached posteriorly immediately below the orifice of the ureters. To-day the cystoscope would have discovered such a septum and led to an appropriate operation.

Radical operation with complete removal of the urachus was successfully accomplished by C. B. Waller[59] from a healthy boy of 5 months, with a navel tumor through which water constantly discharged. At the umbilicus there was a red, inflamed, and very sensitive tumor. Under chloroform a catheter passed with ease into the bladder. An incision was made through the skin surrounding the tumor and the urachus dissected out for an inch, ligated with stout silk, and the end allowed to fall into the abdominal cavity (?). The edges of the abdominal wound were brought together by a quilted suture and superficial silver wire. The child was discharged on the twenty-first day, urinating normally and cured.

Stierlin[60] in more up-to-date technic, excised the tumor and 1½ inch of the urachus down to the bladder vault, tied off with catgut, sewed the wound, which healed in ten days. Stierlin maintains that operation is the only rational treatment for these cases.

Mikulicz[61] operating upon a boy of 5 months made a circumferential incision around the umbilicus, dissected out the urachus down to the bladder, cut off the urachus, closed the bladder and abdomen by suture, and the child did well.

Freer[62] "emphasizes the importance of examining carefully the urethra before proceeding to operate for closure of the fistula." The diagnosis of this abnormity may seem too simple to demand remark, and, indeed, it is simple when complete patency exists, but when it is only partial, and the tube undilated, or, when it is much dilated,

[58] Trans. Acad. Med. Ire., '83–4, ii, 373.
[59] Med. Bull., Phila., '85, vii, 371.
[60] Loc. cit.
[61] A. Jahu, Beitrage klin. Chirg., 1900, No. 26 hft. 2.
[62] Loc. cit., p. 114.

the diagnosis becomes much more difficult, and its true nature often escapes recognition by the most skilled diagnostician. The following characteristics may be of some service where there is danger of confounding it with other abnormal growths in this region:

"When a cyst is attached in the pelvis in the median line and grows gradually from this attachment upward toward the umbilicus, where it also has attachment, the uterus being depressed, it may be held to be a cyst of the urachus.

"If the cyst be then punctured and the withdrawn fluid found to contain flattened epithelium along with urea, and the other urinary salts, the diagnosis is practically certain."

Urachal Dilatations.—The urachus may be found dilated at or even before birth, at its vesicle end, its umbilical extremity, or in one or several portions throughout its length.

"Cysts of the urachus are congenital or acquired. The acquired cyst may, so far as I have seen, arise at any period of life, up to 45 years of age, and I know of no reason why it cannot occur at any age. It appears that the unclosed cavity in the urachus may remain dormant for years, and then take on a pathologic condition. Any part of the urachus may dilate, but the part next the bladder seems most liable, as that is the last portion to become functionless." [63]

Robinson declares that the frequency of cystic dilatation of the urachus is difficult to estimate. During his examination of 40 sheep, 60 cows, 175 dogs, no trace of urachal dilatation was found; and in 150 sows not one as large as a bean was seen. But it was quite a different matter in the fetal pig, of which he examined about 30. In the very early fetus the bladder was elongated, and it was frequent to see cysts which one could easily record as urachal cysts, but no doubt they rapidly closed in later fetal life. During several years' work in the dissecting room, he saw but one urachal cyst (Fig. 12) but many variations in the appearance of the urachal cord. Meckle and Hoffman each report urachal cysts in pigs; J. Bland Sutton has seen a urachal cyst in a mole; both Gurlt and Freer give excellent examples of urachal cysts in the horse.

In 1530, Cabrol described a urinary fistula in a young girl,

[63] F. B. Robinson, Annals of Surgery, 1891, xiv, 337–355.

detailed its relations, and suggested treatment.[64] It is to J. B. Wutz [65] that we owe our present knowledge of the frequency of the presence of small urachal dilatations which he found present in 24 out of 74 post-mortem examinations, varying in size from microscopic dimensions to the size of a bean. They all lay in the lower third of the urachus; were lined with pavement epithelium, as a rule stratified, and there was a capsule of plain muscle cells on the outer surface, a very important fact in relation to the microscopical examination of sections of the wall of the cyst suspected to be urachal. These "Allontoic cysts" of Lawson Tait, present as one or more sacs along the course of the urachus, are aptly likened by Douglas [66] to "a string of sausage," or by Ill as presenting the appearances as if it were knotted or had excrescences." [67]

A congenital allantoic cyst in a still-born female has been described by W. L. Ransom.[68] A tumor at the umbilicus the size of a child's head, which was a portion of a multilocular cyst springing from the pelvis, attached by three large fibrous bands and consisted of one small and three large cysts, filled with urine. Heart and liver greatly enlarged, lungs undeveloped.

Small urachal cysts may remain throughout life without giving any symptoms, as shown by the work of Wutz and others who found them post-mortem and during operation for other purposes.

Large Urachal Cysts.—Cysts which do not open at either the vesicle or umbilical end often attain enormous proportions, become manifest according to Douglas between the ages of 5 and 66 years; are more common in the female (8 males to 27 females) and are more common in boys than girls.

"All cases of urachral cysts do not present pronounced symptoms. . . . Many behave very innocently, occasioning only moderate distress until they attain quite a growth. As they develop they extend down into the pelvis, and displace the intestines and peritoneum upward, and the pelvic viscera downward (Fig. 14). Under these conditions cysts may simulate ovarian or parovarian

[64] Alphabet, Anat. Obs., No. 20, 1550.

[65] Ueber Urachus und Urachuscysten. Arch f. path. Anat., Berlin, 1883, xcii, 387–423.

[66] American Journ. Obstet., 1897, xxxvi, 474.

[67] Trans. American Assoc. of Obstet. and Gynecol., 1892, v.

[68] Medical News, 1893, lxiii, 547.

cyst, tubercular peritonitis, ascites, abdominal pregnancy, etc. Thornton [69] operated upon what he designates "A Peritoneal Cyst with Blood Cyst of the left Ovary." On opening into the cyst he could not at first be sure that he had opened the peritoneum. After tapping the cyst and shelling out its sac, the cavity passed into the pelvis, and the uterus could be recognized through a thick membrane. He then recognized another cyst within the abdomen, a hematoma, which with the ovary was removed, without having entered the peritoneal cavity.

In corroboration of the antenatal causation of urachal cysts Freer [70] relates the following of a divinity student, H. R., who had from infancy been remarkable for a large abdomen, which made him the subject of much ridicule among his playmates. A puncture was made, penetrating the walls of the fluctuating mass. This was followed by vomiting and intense abdominal pain. A considerable quantity of reddish-yellow fluid was discharged. This measure gave relief for two years. A puncture was again made and about 6 liters of fluid withdrawn. This operation was repeated three times during the nine months, the remainder of his life. At death he weighed 192 pounds, the fluid in the cyst amounting to about 100 pounds.[71]

In the department of urachal cysts Mr. Lawson Tait has been a real epoch maker. What he has taught in the past eight years about these cysts has been a revelation to abdominal surgeons.[72]

In 1886, Tait presented before the British Gynecological Society a report of "Twelve Cases of Extraperitoneal Cysts," [73] to which others have since added their experience, and as the result of his investigations and experience with one case of a large cyst from which he removed 25 pints of fluid [74] concludes with the following advice as to treatment: "Incision into the cyst, thorough evacuation of its contents, irrigation of the cyst cavity, mopping out with a solution of iodine, and circular drainage through Douglas' cul-de-sac in females, and tubular drainage in males, now appears to be the chosen method of treatment by Mr. Tait; and since he declares, and

[69] Trans. Path. Soc., 1878, xxix, 145.
[70] Loc. cit. p. 111.
[71] Rippmann, Deutsche klin., '70, xxii, 267.
[72] F. B. Robinson, Ann. Surg., xiv, 1891, 343.
[73] Brit. Gyn. Journ., 1887, ii, 328.
[74] Am. Journ. Obstet., 1897, xxxvi, 482.

no doubt correctly, that there are many of these cysts which dip down into the pelvis that cannot be removed, we are forced to adopt as a *dernier ressort* something like the above plan of treatment. The more radical operation of enucleation of the sac, when it is possible, has a finish (literally so), a completeness, about it attractive to the surgeon. The only objection I can see to it was sorely impressed upon me by the fatal result of my case—it is the separation from the parietes and devitalization of the peritoneum. If we remove these cysts what is to be done with the peritoneum? Mr. Tait suggests the probable propriety of stitching the peritoneum back to the parietes. I seriously considered this in my operation, but concluded it would accomplish but little.

"Were I to encounter this condition again I should adopt one of two methods: First, excise the detached peritoneum, spread the omentum over the fresh surface and attach it, thus lessening very greatly the danger of intestinal adhesion and subsequent obstruction; or, second, affix the omentum to the detached peritoneum and stitch them both to the parietes, thus relying upon the omentum to assist in maintaining the vitality of the peritoneum."

Vesico-Urachal Dilatations.—Douglas[75] says "that a cystic expansion of the vesicle end of the bladder urachus communicating with the bladder is not a true cyst, but, as designated by Schuellenbach, a cystic ectasis," or vesicle diverticulum.

"A dilated urachus, accidentally opened while performing abdominal section," occurred in the hands of G. Morgan[76] on a boy of 5 years; the catheter passed through the bladder and cyst to the umbilicus. There were evidences of recent peritonitis. The child was too ill to permit dissection of the cyst, which was washed out, and closed by a double row of silk sutures. Pus present on the third day; the wound had healed and the boy was well on the twenty-sixth day. Retention was supposed to have been caused by distension of the intestines.

Scholz[77] saw a girl, aged 16, who had had a tumor at the navel for 8 weeks, and suffered from severe pain in the abdomen, and dysuria. After one month's symptomatic treatment, a hair-like

[75] Loc. cit.

[76] Lancet, Lond., 1896, ii, 1154.

[77] Wien. med. Woch., 1878, xxviii, 1327.

opening appeared at the navel and urine escaped. Scholz enlarged this opening, inserted a drain, and she recovered in four months from the time when she was first seen.

The progress of a vesicle diverticulum is well shown by F. Bramann's [78] story of a girl, aged 12 years, who since her ninth year had suffered with pain in the abdomen, chills, and fever, followed by frequent urination and the passage of milky and purulent fluid. For a time the symptoms subsided, but one year later fluid began to pass through the navel, clear or flaky and of a urinous odor. A permanent catheter in the urethra proved useless; the cyst was split up and curetted; the bladder opening closed; the wound partially sutured and in part packed, and the girl was cured.

The Recognition of Vesicle Diverticulum.—According to Douglas [79] "the patency of the vesicle end of the urachus is very truly an abnormality, an arrest in the general obliterative process by which many functionless tubes become cords and ligaments, and it will perhaps escape observation unless some condition arises preventing the free flow of urine from the bladder; then it is that a series of phenomena appear to engage the attention of the surgeon. The clinical picture of these cases may be briefly stated as follows: Usually there exists in an acute or chronic form some pronounced disease of the urethra or bladder in which has occurred retention of urine or great vesicle tenesmus. Suddenly there develops a tumor just above the pubes, at first of small size, distinctly circumscribed, quite hard, too tense to be fluctuant, and sometimes very tender. There is marked diminution in the amount of urine, and usually great dysuria. The only case in which I can find this symptom (dysuria) absent was one carefully recorded by Freer. Constitutional symptoms, nausea, vomiting, and great depression are noted. Pain all over the lower abdomen is a constant symptom. Gradually the tumor enlarges, becomes softer, more elastic and fluctuant. The local signs and general symptoms depend entirely upon the character of the urine that is forced backward into the urachus. If it is septic, soon we have a purulent inflammation of the walls of the urachus and the hypogastric tumor may closely resemble an acute abscess. There is, however, a frequent and more

[78] Arch f. Klin. Chirg., Berlin, 1887, xxxiv, 1001.

[79] Loc. cit.

fortunate termination for these cases having vesicle connection. By catheterization or otherwise the distended bladder is relieved, the urachus is drained into the bladder, the tumor suddenly disappears, and all pain and symptoms quickly subside."

Unterberger[80] gives a very clear account of a similar case of urachal fistula, caused by a retroflexed, pregnant uterus in the fourth month of gestation. The abdomen was swollen; almost entire failure to urinate; the bladder enormously distended. It was not thought wise to replace the womb immediately, nor interfere with the fistula. A catheter was inserted in the bladder, and in a few days the discharge through the umbilicus ceased, and the uterus had risen from the pelvis, and it was finally possible to bring the uterus to the normal by tampons. Abortion occurred later. The bladder was not infected in spite of the extraordinary retention.

Umbilico-Urachal Sinuses.—Whenever a urachal dilatation which does not extend to the bladder, ruptures at the umbilicus, it differs from a vitelline sinus in the nature of its secretion as illustrated by Van der Veer's[81] case of "Congenital Sinus of the Urachus." An unusual discharge from the navel was discovered by the nurse, ten days after birth. When 21 years old, the patient complained of a very distressing, profuse and very offensive discharge through the umbilicus, also an unpleasant sickening sensation so that she was unable to bear the weight of her clothing and was prevented from horseback riding or any other active exercise. From the parts above the navel there exuded at times a *sebaceous substance* quite sticky, and very offensive in odor, which excoriated the parts somewhat. A probe readily passed some three inches toward the fundus of the bladder. Excision was followed by recovery.

A sebaceous concretion was removed from an urachal sinus, by F. R. Walters,[82] from a man 34 years old, who had suffered from severe periodic attacks of colic. The pocket was 2½ inches in diameter, ¾ inch deep, with an opening ½ inch; no hernia. The

[80] Monat. f. Geburt. u. Gynakol., 1900, Bd. xi, H. 3.
[81] Trans. Amer. Assn., Obst. & Gyn., Phila., 1889, ii, 7.
[82] Brit. Med. Journ., Lond., 1893, i, 173.
[83] Todd's Cycloped. Anat, and Physiol., 1835–36, i, 393.
[84] Lehrb. der path. Anat., 1861, iii, aufl. iii, 372.

mass was made up of sebaceous material and the "hairs" from the flannel belt worn by the patient.

In Boyer's [83] case from a sinus 1½ inches deep, he removed 12 urinary calculi, each the size of a millet seed, and Rokitansky[84] found 21 stones the size of milium seed 6 lines above the urachus capsule.

SUMMARY OF TREATMENT OF URACHAL FISTULAS, SINUSES, AND SMALL CYSTS.

In a paper read before the American Surgical Association, July, 1905, G. T. Vaughn briefly reviewed 52 cases of "Patent Urachus" reported in the literature and added one of his own; of this number 38 were operated on, with 32 recoveries and 3 deaths, and in three cases the result was not reported. He divided the methods which have been tried for closing the urachus into eight classes, as follows: (1) The application of caustic or the actual cautery to the umbilical opening. (2) The use of the cautery and of ligatures or sutures. (3) The use of ligature or suture only. (4) Plastic operations, the application of a flap of skin to close the opening. (5) To open or to slit up the urachus, curette or cauterize and pack. (6) Removal of the sources of irritation from the urachus, such as stones, pus, etc., and keeping the parts clean. (7) Removal of the obstruction to the flow of urine from the bladder through the urethra, as a tight prepuce, strictures of the urethra, hypertrophy of the prostrate, and tumors or stone in the bladder. (8) Extirpation or dissecting out of the urachus and sewing or ligating the part next the bladder, after the manner of treating the appendix in appendectomy. Dr. Vaughn does not approve of the method of simply closing the umbilical end of the fistula, lest an infected suppurating area be left which may cause septicemia or peritonitis. He considers slitting open and packing the abscess cavity a rational operation, but thinks the operation of election is to dissect out the urachus with the entire diseased area. Dr. Vaughn used this method in his case, a man of 40, who had had a patent urachus since the age of seventeen. A month after the patent urachus was cured, it was necessary to perform nephrolithotomy on the right kidney, and nine months later the same operation on the left kidney.[85] This case, truly, is a powerful argument in favor of early, radical operation.

[85] Society Report Journ. American Med. Assn., 1905, xlv, 347.

Hepatico-Urachal Fistula.—An unique case is recorded by Kostlen, Bramann,[86] of a female, aged 38 years, who passed gallstones and bile with the urine. Autopsy 2 years later, showed a duct leading from the gall bladder direct to the urinary bladder.

Gall Bladder Fistula.—Bramann[87] relates the history of a female, 63 years old, who when 45 noticed a tumor at the navel, which disappeared four weeks later. After two years the tumor reappeared, opened at the umbilicus, discharged foul, purulent fluid. This was split open, scraped, drained and healed in three months. Several gallstones were removed from the sac. Von Bergmann treated a similar case in the same way.

RESULTS OF OPERATIVE TREATMENT OF LARGE URACHAL CYSTS

Cases.	Method.	Operator.	Rec.	Died.	Where Found.
1	Repeated tapping.	Rippmann.		1	Deutsche Klin., 1870, xxii, 267.
2	Repeated tapping.	Hoffmann.		2	Archiv f. Heilk., 1879, xi, 375.
2	Enucleation sac.	Tait.		2	Brit. Gyn. J., 1887, ii, 328.
1	Enucleation sac.	R. Park.	1		Caroll, Buffalo M. J., 1895-6, xxxv, 869.
1	Enucleation sac.	Douglas.		1	Am. J. Obstet., 1897, xxxvi, 477.
1	Incision and Drainage.	Heinecke.		1	Handb. d. Allg. u. Spec. Chir. 3, 2, 2, 64, 1879 (Supra) and Robinson, Ann. Surg., xiv, 350. Teichelmann, Lancet, 1888, ii, 675.
15	Incision and Drainage.	Tait, L.	13	2	
1	Incision and Drainage.	Helmuth.		1	Freer, Ann. Surg., '87, v, 116.
1	Incision and Drainage.	Pratt & Bond.	1		Lancet, Lond., 1890, i, 896.
1	Incision and Drainage.	A Colleague.	1		Ann. Surg., 1891, xiv, 353.
26			16	10	

SUMMARY:

Repeated tapping.....	3 cases.	Rec.,	0.	Died,	3.	Mortality 100 per cent.
Enucleation sac	4 "	"	1.	"	3.	" 75 per cent.
Incision and drainage.	19 "	"	15.	"	4.	" 21 per cent.

[86] Arch. f. klin. Chir., Berl. '87, xxiv, 1009.

[87] Loc. cit. p. 1004.

Obstetrics and Gynecology

TUBAL PREGNANCY TERMINATING IN SPONTANEOUS RECOVERY WITHOUT OPERATION; EXHIBITION OF A SPECIMEN OF RUPTURED TUBAL PREGNANCY; OVARIAN ABSCESSES AND TUBAL ABSCESSES; RIGHT-SIDED HYDROSALPINX, LEFT-SIDED HYDROSALPINX, AND LEFT-SIDED OVARIAN CYST, COMPLICATING A FIBROID TUMOR OF THE UTERUS

A CLINICAL LECTURE DELIVERED AT THE WESLEY HOSPITAL, CHICAGO

BY THOMAS J. WATKINS, M.D.

Clinical Professor of Gynecology in the Northwestern University Medical School; Gynecologist to St. Luke's, Mercy, and Wesley Hospitals, Chicago, Illinois

TUBAL PREGNANCY TERMINATING IN SPONTANEOUS RECOVERY WITHOUT OPERATION

GENTLEMEN: This case (Case I) is more interesting than many an operative case. The diagnosis is tubal pregnancy on the left side. This patient has been very unfortunate, in that she was admitted to Wesley Hospital to my care last April with a tubal pregnancy upon the right side, and the right tube was removed. The appendix was adherent, and was also removed. There were numerous peritoneal adhesions at that time, due to the fact that she had been curetted two or three times before admission to the hospital for a hemorrhage from the uterus, which was supposed to be due to a miscarriage. Last August she was again admitted to the hospital to my care with intestinal obstruction, and on section it was found that a small band of adhesion extended across a loop of the small intestine, producing almost complete obstruction. Her general condition was very bad at that time, but she made a very rapid recovery and

left the hospital in good health between the second and third weeks.

She was taken ill about two weeks ago, and with this illness she had much pain, which was located in the region of the left tube. This pain was intermittent in character. (Intermittent pain is a valuable diagnostic symptom between ectopic pregnancy and salpingitis.) She felt as if she would faint at times, which is often a valuable diagnostic symptom in ectopic pregnancy. The woman says she felt at this time exactly as she felt last April, with the exception that at this time the symptoms were referred to the left side, while last April they were referred to the right side. Menstruations at this time were not interrupted. She menstruated one day ahead of time. In April the menstruations were interrupted for about two weeks. As a rule, menstruation is delayed with tubal pregnancy, but there are numerous exceptions to the rule, and it is very probable that in those exceptional cases the woman menstruates at the regular time with the pregnancy in the tube. We take it that probably this woman had her last menstruation while pregnant in the tube.

On conjoined palpation a mass was found in the region of the left tube, two or three inches in diameter. This mass is of recent origin, because the patient had been under observation only a short time ago. This mass is not as sensitive as is usual in an inflammatory swelling. The disease is unattended by any elevation of temperature, which would not usually obtain if it were an inflammatory mass. The sudden appearance of the mass would exclude a neoplasm. If one excludes a neoplasm then it is necessary only to distinguish between an inflammatory swelling and a tubal pregnancy to make a diagnosis. We can probably exclude an inflammatory swelling here by the absence of any history of infection, by the absence of fever, leukocytosis, and tenderness. This patient was under observation a short time ago, and we believe it is quite certain that she has not been infected since then. The character of the pains were those of tubal pregnancy. They were intermittent, not continuous. There was no elevation of temperature. There was marked menstrual disturbance at this time. The patient has been flowing constantly for about two weeks, dating from the menstruation, which is uncommon in inflammatory cases.

The patient was brought to the hospital with the idea that she

would probably have to be operated upon. She was kept under observation, however, for a short time. While under observation the treatment consisted in keeping her quiet and keeping an ice-bag over the seat of pain; the use of sufficient codein to relieve pain, regulation of the diet, and the administration of mild cathartics.

Examination yesterday showed that the mass has diminished one-half in the last four or five days; the pain has diminished materially, and the menstrual discharge has almost disappeared. The flow is scant to-day. The diagnosis in this case is not only tubal pregnancy, but a tubal abortion. We believe that this tube has aborted into the abdominal cavity, and that the abortion is complete. We know that absorption is taking place, because the mass has become much smaller, and in all probability this case will go on to a complete spontaneous recovery. It is necessary, however, to keep the patient under observation as long as there is any pain, and as long as the swelling persists.[1]

This case is not an exceptional one. There is no doubt that a very large percentage of cases of ectopic pregnancy terminate in spontaneous and complete recovery, and that most of these cases are probably not diagnosed. Some of them are diagnosed as cases of salpingitis, "inflammation of the bowels," and the like. It is, however, possibly somewhat dangerous to give this favorable prognosis of tubal pregnancies without any operative treatment, on account of the danger of neglect in some cases that would rupture and possibly terminate fatally without early operation. The rule has become general, and I believe it is a most conservative treatment, to operate on tubal pregnancies as soon as they are diagnosed, or as soon as satisfactory preparations can be made after the diagnosis has been determined. This is the second case I have seen within a year, in which there is very little doubt that tubal pregnancy existed, tubo-abdominal abortion resulted, and the patients recovered without operative treatment.

EXHIBITION OF SPECIMEN OF RUPTURED TUBAL PREGNANCY

CASE II.—The patient from which this specimen was removed was sent to the hospital with a diagnosis of general peritonitis and died within an hour after admission. The specimen, Fig. 1, consists

[1] This patient made a complete spontaneous recovery.

Fig. 1.—Ruptured tubal pregnancy.

the tube, products of pregnancy, blood-clots, adhesions, and ovary. The size of the fetus would indicate an eight to ten weeks gestation. The chorionic villi are beautifully illustrated. The pregnancy occurred in the ampullar portion of the tube and an extensive rupture took place near the fimbriated end. Judging from the size of the fetus it is probable that slight ruptures and hemorrhages took place before the severe attack occurred. This specimen is a beautiful illustration of ruptured tubal pregnancy, emphasizes the dangers of tubal pregnancy, and gives an opportunity of impressing upon you the difficulty of diagnosis. Typical cases are easily distinguished when looked for, but atypical cases are numerous and some of these cases are among the most difficult to diagnose in gynecology. This specimen was removed on necropsy (Fig.1).

OVARIAN ABSCESS AND TUBAL ABSCESS

Case III.—This woman is twenty-eight years of age, single, a Jewess, and weighs 116 pounds. She has lost considerable weight in the last few weeks. The family history states that her father died of Bright's disease; her mother is living and well; three sisters and three brothers are living and well. As to previous illnesses and treatment, she has had the diseases of childhood. There is no specific history. Five weeks ago the uterus was curetted, but the operation gave no relief. The patient now complains of severe pains in the back and in both groins, and tenderness on the right side. The pain is referred down the thigh, especially along the anterior crural nerve.

The history of her present illness dates back five weeks, at which time she had gone six weeks without menstruating. She was then taken with sharp, severe pains in the back and groins, and the following day began flowing. The amount of the flow was more than usual at the menstrual periods. The patient says that she flowed continuously for three days, when she was taken to a hospital and curetted. She flowed slightly for the two days following. These pains have continued ever since, and the operation gave her no relief. Her menses began at fourteen, and is of the regular twenty-eight day type; duration, three days; always regular before this. Prior to this illness the date of her last menstruation was

September 27. Vaginal discharge was whitish before and after periods. As to vesical symptoms, she has no trouble with frequent urination; no smarting. Appetite was fair. She is sometimes sleepy, particularly after meals; her bowels are constipated.

Examination on October 23 showed the perineum was relaxed, the cervix slightly dilated, the uterus slightly enlarged, and in normal position. A globular mass posterior and to the right of the cervix, about three inches in diameter, and very sensitive to pressure, was found. The left appendage showed slight thickening. Vaginal canal contained a whitish discharge. The right kidney was palpable and normal. On October 25, the mass was of the same size. On October 24, the history sheet states that at 8 A.M. the temperature was 98.8°F; pulse 84; respirations 20. At 4 P.M. the temperature was 97.4°F; pulse 84; respirations 20. October 23, the temperature was 98.1°F; pulse 84; respirations 18. As to blood findings, leucocytes were 14,400. Differential count showed 10 per cent. lymphocytes, 20 per cent. large mononuclears, and transitional, and 70 per cent. neutrophiles. The urine was normal.

The diagnosis in this case was somewhat uncertain. The indications were a tubal pregnancy, but we could not make an absolute diagnosis of that condition. I take it that this case belongs to those in which there may be a suspicion of tubal pregnancy, but in which it is absolutely impossible to make a positive diagnosis without an exploratory incision.

The abdominal incision is now complete and I will describe the findings.

First, we find numerous adhesions. There is also a somewhat enlarged right Fallopian tube open at the abdominal end and a very large right ovary. The ovary is about three inches in diameter and is embedded in adhesions. These adhesions are fortunately easily separated without producing any rupture of the ovary. The swelling is rather large, and is difficult to deliver through this opening without rupture. The ovary is now delivered without rupture and so far as the diagnosis is concerned we can exclude tubal pregnancy, but there is still a possibility that we have an ovarian pregnancy. It is well to remind you that there is no question that ovarian pregnancy does occasionally occur. I now grasp the broad ligament by passing a forceps down over the infundibulo-pelvic

ligament, which when clamped shuts off the blood-supply from the ovarian vessels. Next, I pass a forceps down over the horn of the uterus, grasping that portion of the broad ligament that contains the uterine artery, so that the blood-supply is now under control, and the ovary and tube can now be excised without danger or bleeding. I am cutting the broad ligament between the tumor and the forceps. The tumor and the entire tube, including the interstitial portion of the tube, are now removed. This is entirely a dry wound, demonstrating that hemorrhage is completely controlled by the two forceps placed as I have described. The left appendage is now brought into view, and exposes an ovary twice its normal size which contains a cyst of considerable size. It is a follicular abscess about one-half inch in diameter, as shown in frontispiece. The temperature and blood findings indicate that this pus is sterile or very nearly so. I now take a piece of dry gauze and simply wipe away the abscess wall. This is an illustration of an ovarian abscess without any tubal infection. This means that the infection gained entrance through the blood-vessels, or through the lymphatics, and it is, in all probability, secondary to a septic endometritis.

Here is another small cyst in this ovary which contains clear fluid. This ovary will not be removed, although I realize that I take some chances in leaving an ovary that contained an abscess. On the other hand, it was absolutely necessary to remove the right ovary, and in a young woman of this age there is great objection to removing both ovaries, unless one is absolutely obliged to do so. It is better to leave a portion of this ovary and run the risk of suppuration occurring in it than it is to remove it and thus bring on the menopause. The greater part of the wound in the ovary is now closed by a mattress suture, and some very fine sutures accurately complete the closure of the wound. Finding an abscess in this ovary makes it probable that the other ovary also contains an abscess. I find the appendix drooping down over the brim of the pelvis; it is very easily brought into view. I shall remove this appendix, as it has a suspicious appearance, and it is safer to remove it than to leave it. First, I clamp the meso-appendix with an eight-inch forceps; second, I make a cuff chiefly of the peritoneum, and this cuff is easily wiped down by the use of a little dry gauze. I crush the appendix at its insertion into the bowel with

an eight-inch forceps, and then ligate with small fine catgut and cut the appendix through the crushed portion. The object of this crushing is to avoid having very much mucous membrane in the part that is tied and cut. In order to guard against infection escaping while we cut, I place a little gauze underneath the appendix on my finger, and with a scalpel cut through the appendix upon the gauze. As soon as I cut my assistant touches it with 95 per cent. carbolic acid and alcohol. The meso-appendix is now tied with a fine ligature. A purse-string suture is run about the base of the cecum and the stump of the appendix is inverted. My assistant, Dr. Gilmore, will close the wound in the right broad ligament, and that closure will be based upon three principles. First, to control the bleeding; second, to cover all raw surface; third, to shorten the broad ligament so as to restore the support to the uterus. The abdominal wound is closed in three layers, using, first, fine catgut for the peritoneum; second, chromicized catgut No 2 for the fascia; third, very fine catgut for the skin, and then the abdominal wound is seated with collodion and covered with a plain gauze dressing. We have given up the use of very thick abdominal dressings because they are uncomfortable to the patient; they are usually too hot and do no good. Furthermore, they are expensive, and the amount of dressings that is often used in hospitals is unnecessarily expensive.

Demonstration of Right Fallopian Tube and Ovary.—The tube is slightly thickened, and slightly injected, but contains no abdominal secretion. The fimbriated end is not adherent, and not closed. You will notice that the tumor mass is composed entirely of the ovary. The ovary is globular and is two or three inches in diameter. It was entirely embedded in adhesions. We will open it to see the character of the contents. It is an abscess. I will not lay it open, because I am anxious to have a smear and culture made of this pus.[2]

This case is an exceptional one of ovarian abscess, especially on account of the very mild symptoms which the patient had. The patient came in with a double ovarian abscess without any elevation of temperature, and, so far as the history goes, the abscess was

[2] Smear and culture showed sterile pus.

recent. It would not be surprising to have a patient come in with a chronic ovarian abscess, with normal temperature.

The conclusions in this case are: This patient was probably pregnant, and she had a miscarriage produced, and thus infection was carried into the uterus, producing a septic endometritis. Uterine hemorrhages occurred, she was admitted to a hospital, and curetted for an incomplete abortion. There was no fever at that time. Dr. Fortner, who referred the patient to me, did not see her before she was curetted, so that we are not certain as to her history as regards the temperature at the time she was curetted. At any rate, the curettage did not stop the pain. Pain increased after the curettage, and the reason for that increase may have been first, the curettage produced abrasions inside the uterus, consequently increased the amount of absorption of septic material from the uterus; second, the curettage may have set free a thrombus in a blood-vessel, and this may have traveled to the ovary and produced a thrombosis in the ovary, which later suppurated and produced abscesses. An embolic infection in the ovaries may have antedated the curettage.

You might ask with a great deal of propriety why this patient did not have considerable fever; why she did not have chills and fever? And the reply to this is, first, the infection must have been of septic matter, but we now know that when there is an absence of fever it means a low virulence of infection, an absence of that the absence of temperature was due to the fact that the pus was well "walled off;" that the patient was not getting any absorption of septic matter, but we now know that when there is an absence of fever it means a low virulence of infection, an absence of bacteria or a high degree of resistance to infection. Clinically, judging from the temperature chart, and the white blood count, we would expect this pus not to contain bacteria. But judging from the recent infection we should hardly think that the bacteria had entirely disappeared from this pus, and it is impossible to arrive at any satisfactory opinion as to whether there are bacteria in this pus or not, without a bacteriologic examination.

This is a good opportunity to speak to you briefly about the difference in origin between ovarian abscesses and tubal abscesses. You have seen numerous cases in this clinic of infection in the tubes, and you have been told in those cases that the disease was an exten-

sion of the infection along the mucous membrane. First, a vulvitis, vaginitis, or endocervicitis; second, an endometritis; and, third, a salpingitis. Here is an infection in the ovaries without tubal involvement, consequently this infection has not traveled by continuity of tissue. This case here follows the general rule that in puerperal cases the infection more often extends through the vessels than it does by continuity of tissue, which is in contrast to the non-puerperal state, in which the infection nearly always travels by continuity of tissue and not through the vessels. We have another law illustrated in this case, and that is, infection of the ovaries is much more common than infection of the tubes in puerperal cases. Infection traveling along the vessels tends to go to the ovaries rather than to the tubes, because the relation of the vessels of the ovary to the uterus is more intimate than the relation of the vessels of the tube to the uterus.

The prognosis as regards life in a case like this is excellent, as the infective material in the ovaries has little or no virulence. What interests us most in the prognosis in this case is the outcome of the left ovary which was resected and which contained an abscess. The danger attached to this case is that possibly some of the Graafian follicles in the portion of the ovary that was left may be infected, and that the pathologic changes were so slight as to have escaped detection. If that is true, there is some danger of her having more suppuration of that ovary, which may require a subsequent operation.[3] But in a young girl it is extremely important to preserve the function of menstruation and reproduction, and it is very much better to take the chances of subsequent trouble developing in that ovary than to produce the menopause and render the woman sterile.

RIGHT-SIDED HYDROSALPINX, LEFT-SIDED HYDROSALPINX, LEFT-SIDED OVARIAN CYST, COMPLICATING FIBROID TUMOR OF THE UTERUS

Case IV.—In the next case I will give you the diagnosis before reading the history, so as better to bring out some of the more

[3] Recovery was uneventful and examination three weeks after operation showed no evidence of left ovaritis.

important points in the history. The diagnosis is uterine myofibroma, which extends up to the umbilicus, and is probably complicated by an old salpingitis or cystic disease of the ovaries and tubes.

The patient is forty-eight years of age, married, and is a housewife. She thinks that she has lost some weight in the last two months. The special object of going into the patient's weight is on account of the possibilities of malignancy. With a large malignant tumor, the patient will probably have lost considerable in weight, and in some cases in which there has been a history of septicemia there is also loss of weight. So the fact that this patient has not lost much weight suggests that the tumor is probably not malignant. However, this does not entirely exclude malignancy, and suggests that it is not due to an acute infection.

With regard to the family history, her mother is living and well; her father is dead, but the cause of death is unknown. She has two brothers and three sisters living and well. One sister died in childbirth. The family history in gynecologic cases is usually not very valuable, and generally is only important in reference to malignant disease.

As to previous illnesses and treatment, this patient had some lung trouble when seven years old, but she does not know the nature of that illness. She had typhoid fever fifteen years ago; otherwise she has been well, so that this disease probably has no relation to any of the previous illnesses.

The patient seeks relief for (1) a large tumor in the right iliac region; (2) considerable pain and some tenderness from the tumor. Here we have a rather large tumor, with very few symptoms. The history of the present illness dates from two months ago, when the patient was taken with a sudden pain in the right inguinal region, about three inches above the middle of Poupart's ligament. This pain lasted about two days, and then passed away. The attacks of pain have occurred once a week for the last two months, and have lasted about two days. The pain radiates down the anterior portion of the thigh and to the back. The patient thinks this tumor has grown since she first noticed it, two months ago. She has pain at present in the right inguinal region.

The fact that we get a history of a tumor dating back two months does not mean that this woman did not have a tumor before that

date. It is surprising what large tumors patients can have without detecting them. Just what these intermittent pains in the right side have been due to is rather uncertain. They may be due to old adhesions from an old salpingitis; they may be due to a catarrhal appendicitis, and they may be urethral in origin. They are probably not due to the tumor itself, because fibroid tumors in themselves do not cause much pain except by pressure, and pressure pains would not be intermittent as these have been.

Menstruation began at fourteen. She has the twenty-eight day type. It usually lasts one week. She has slight pain at the first part of menstruation. She is flowing at present, menstruation having begun five days ago. She has had seven children. The children were born between the years 1876 and 1884. Her labors and recoveries have been normal, so that this disease probably bears no relation to her former pregnancies and labors. She has had no miscarriages. There has been no vaginal discharge. There are no vesical symptoms. The history is negative as to disturbances of the alimentary canal. Physical examination of the heart and lungs shows these organs to be normal.

Abdominal examination reveals a tumor which extends from the pubes upward to the umbilicus. It is irregular in outline, somewhat more prominent on the right than on the left side. There is tenderness in the region of McBurney's point. The tumor is moderately movable and apparently solid. The right kidney is palpable and normal. There is no ascites. The abdominal examination is otherwise negative. Vaginal examination discloses the perineum somewhat relaxed; urethra normal; cervix two-and-a-half inches from the vulva, which is about the normal distance. The cervix is continuous with the tumor. The uterus cannot be made out separately from the tumor, and the cervix and tumor apparently move together. Rectal examination shows external hemorrhoids. Blood examination shows a leukocyte count of 11,000, which is a very slight increase from the normal. The urine was examined, and found normal. The patient's temperature is normal, as is also the pulse.

I will give you very briefly an outline of the practical points as regards fibroid tumors of the uterus. *Etiology.*—There is very little known about the etiology of fibroid tumors of the uterus.

There has been a good deal written about the etiology, and you know it is possible to say a great deal about things of which little is known, and that applies to the etiology of fibroid tumors of the uterus. They never occur before puberty. They are rather uncommon in women under twenty or twenty-five years of age, and they probably never commence after the menopause. They may diminish in size but do not continue to grow in the absence of degenerative changes after the menopause.

As to the *pathology* of fibroids, they are always confined in a capsule. They are generally multiple. Their size varies from those microscopic in size to tumors that weigh fifteen or even one hundred pounds. They receive their blood-supply from the capsule. In color they vary according to whether they contain a large amount of connective tissue or whether they contain a large amount of muscular tissue. They are hard or soft, in accordance with whether they are chiefly fibrous or whether they are chiefly muscular, and it is not uncommon for them to be soft as the result of edema. They are often edematous. They are subject to degenerative changes which often affect their color and consistence. They occasionally become cystic. They may become carcinomatous or sarcomatous,—likely more sarcomatous than carcinomatous, because they originate in tissues which favor sarcoma rather than carcinoma. They quite commonly undergo calcareous degeneration.

Subjective Symptoms.—The most common subjective symptom is hemorrhage. The hemorrhages usually occur at the menstrual periods, a circumstance important in distinguishing between fibroids and some other solid tumors of the uterus. These hemorrhages may cause the menstruation to last longer, and these tumors sometimes cause menstruation to occur more frequently than normally. Hemorrhage, however, is not a constant symptom of fibroids of the uterus as in this case. One finds numerous fibroids in some cases without hemorrhage. These patients have hemorrhages usually on account of one or two causes, or of two causes combined: First, an increase in the area of the endometrium—these tumors tend to cause hypertrophy of the endometrium and, second, a fibroid tumor may interfere with the return circulation of blood from the endometrium.

The second subjective symptom is pain, and pain from fibroids

is due to pressure. Pain, however, usually is not present unless the tumor is impacted in the pelvis or unless it is very large. One frequently gets all the symptoms of anemia, depending upon whether a patient bleeds or not, and depending upon the amount of blood lost, and how well the patient tolerates the loss of blood. It is probably well for me to emphasize the personal equation in all diseases, and one should study the patient as well as the disease. For instance, one woman may bleed a great deal from a fibroid tumor, but may show very little constitutional disturbance from the hemorrhage; while another woman may lose the same amount of blood and show very marked constitutional disturbance. Numerous and severe symptoms may occur from complications, especially from infections.

The objective findings are: First, we usually find a tumor which moves with the cervix. The tumor can usually be made out as being continuous with the uterus, and very frequently it is impossible to distinguish between the uterus and the tumor. The tumor may or may not displace the cervix, depending upon its location. There may be various symptoms from complications or fibroids, such as degenerative changes, infections, adhesions, appendicitis, etc.

The diagnosis of fibroid tumors depends, first, upon the clinical history; and, second, upon the presence of the tumor. When one finds a pelvic tumor, he tries to determine whether it is uterine or extra-uterine. Most fibroid tumors are uterine, and if the tumor is uterine one should always exclude pregnancy. Why? It may seem to you that it would be very easy to distinguish between pregnancy and a fibroid tumor, but the fact remains that it is sometimes extremely difficult. Sometimes operators become rather careless, and, at all events, there have been numerous such mistakes made. For instance, pregnancy has been diagnosed as fibroid tumor, and the patient has been subjected to abdominal section. On the other hand, a fibroid tumor has been diagnosed as pregnancy. In distinguishing between fibroid tumors and pregnancy, if there is any doubt, one should always wait, because time will always enable one to distinguish between pregnancy and fibroid tumors, and the fact of the similarity is emphasized by stating that very good operators have been unable, even after the abdomen has been opened, to dis-

tinguish between a pregnancy or a fibroid tumor, on account of the two conditions being at times so similar.

The patient is now ready for operation.

In this case it is necessary to make a large incision in order to deliver the tumor. We cut boldly through the skin until we come down to the fascia; next we cut through the fascia, which opens up the sheath of the rectus muscle on one side; then with scissors we lay the fascia open throughout the length of the wound. Next we split the muscle which exposes the peritoneum. The peritoneum is grasped with two forceps and incised between the forceps. By means of sight we guard against cutting or wounding the bladder. We detect the bladder by the difference in the color of the bladder and the rest of the peritoneum. We have now come down upon a tumor, and the first thing we observe is that the omentum is adherent from one side of the pelvis to the other. These adhesions mean that there has been an infection. The patient gave no history of any puerperal infection, so it is probable that the infection is non-puerperal, and a non-puerperal infection nearly always extends by way of the endometrium to the tubes, consequently she probably had an endometritis and later a salpingitis. I find that the adhesions can be separated without much difficulty. I am now trying to bring out a part of the tumor so that you can see it, but I find it is still held down by adhesions. I am going to open now a small cyst, which is a cyst of the left ovary. Now, you observe, I bring the tumor up, so that it is visible. You see the right broad ligament, which shows that this tumor has developed somewhat into the right broad ligament. I find here a hydrosalpinx on the right side, which accounts for these periodic pains the patient had on the right side. This is a beautiful specimen of hydrosalpinx. It shows you typical adhesions between the omentum and tube. The ovary is rather large, and as the woman is near the menopause, I think it is better to remove it, than run the risk of its giving her trouble afterward by leaving it. We clamp the broad ligament the same as we did in the previous case from above downward, underneath the ovary and tube, and over the infundibulo-pelvic ligament. We then clamp off the supply of the uterine artery at the right horn of the uterus. The broad ligament is now cut between the tumor and the forceps. It is surprising to find that the interstitial and isthmic portions

of the tube are not involved. An eight-inch forceps grasps the broad ligament below the cut surface and includes the uterine artery near the cervix, and the broad ligament is severed between the tumor and the forceps. As it is difficult to get at the left broad ligament, on account of adhesions, we cut through the cervix and separate the left broad ligament from below upward. The cervix is cut through from the posterior to the anterior surface so as to avoid injury to the bladder. The left uterine artery is clamped near the cervix and we find a very complicated mass of adhesions involving the left uterine appendage. We are not surprised that this woman had pain, but the great surprise is that she did not have more pain. I find a loop of intestine which is very firmly adherent, and which we have to separate cautiously for fear of injuring the bowel. We examine the bowel carefully after this separation for fear of injury and find its coats intact. The left ovary contains a cyst about 4 inches in diameter, and is probably the result of an old infection (periovaritis).

Here is a loop of intestine with very firm adhesions to it. We shall close a slight injury which we have made to the serosa of the bowel by a mattress suture. It is difficult to separate the adhesions of the left tube. We have now separated the tube and find a hydrosalpinx; the ovarian cyst is extremely adherent. The adhesions of the ovary are chiefly in Douglas' cul-de-sac. Now we have succeeded in separating them, and we find a very old adherent cyst of the ovary with a hydrosalpinx on top. Here I show you the fibroid tumor of the uterus, with a right-sided hydrosalpinx, a left-sided hydrosalpinx, and a left-sided ovarian cyst.

The first ligature takes the place of the clamp on the right uterine artery and is tied as the forceps is removed. I next tie the portion of the broad ligament which contains the left ovarian artery. These arteries are seen on the cut surface of the broad ligament and are also tied separately. This controls the blood-supply from the left broad ligament. The next step is to control the blood-supply from the right broad ligament, which is done in the same manner. The anterior and posterior flaps of the cervix are now united by a continuous catgut suture. We have to cover a large amount of raw surface in this case on account of the numerous adhesions. A suture is now passed through the infundibulo-pelvic ligament, then

through the round ligament and then through the stump of the cervix on one side, and when tied covers up all of the raw surface on that side and doubles the broad ligament upon itself and thus gives good support to the pelvic flow. This suture is reinforced by one or two sutures and the same is done on the opposite side. The raw surfaces are now all covered, the pelvic floor well supported, and the bleeding stopped. There is no indication for the use of drainage. The wound is closed as in the former case. The after treatment of these cases will be: (1) Codein, if needed for severe pain. (2) Normal saline enemas, one pint every six hours. (3) Water by mouth as soon as desired, preferably hot. (4) Liquid diet after an interval of twenty-four hours. (5) Cathartics are commenced in twenty-four hours. The wound is not disturbed for about two weeks. The patients will be up at the end of the week, or sooner if they desire.

THE USE OF FORCEPS IN DEEP TRANSVERSE ARREST OF THE HEAD

BY CHARLES B. REED, M.D.

of Chicago, Illinois

The arrest of the head in the transverse position after it has descended well into the pelvis is not an uncommon event in obstetric practice, and it is extremely important that an accurate diagnosis of this condition should be made before instruments are used. It happens not unfrequently that a labor is delayed, the bag of waters ruptured, and after three or four hours (usually not so long) the attendant feels that forceps are required, and he applies them and extracts the head with considerable difficulty, if at all, by applying the blades to the sides of the pelvis without regard to the position of the head in the pelvis. This makes the operation unusually difficult if not impossible, and greatly increases the danger to the woman, since if the head does not rotate in its progress, much damage may result.

The operator is not entirely to blame, however, for American obstetric literature is strangely silent in regard to this important position. Hodge described the position very fully, but since his time, no authors except Edgar and Dorland have considered it worthy of particular notice. In the text-books it is habitually classified as a minor occipito-posterior position and described and dismissed as incidental in importance. On account of the definite character of this position, its frequency and the great care which it demands in the use of the forceps it seems entitled to far greater consideration.

This is the first of the deflexion positions, and exhibits the chin somewhat removed from the sternum, the head slightly extended and well down on the pelvic floor. The internal examination reveals the saggital suture parallel or almost parallel with the transverse diameter of the pelvic outlet, the fontanelles being at about the same level on opposite sides of the pelvis, and the labor at a standstill.

There is no tendency toward anterior rotation of the occiput on account of a definite interruption of the rotating mechanism. This is also called "the impacted head" by some writers, and "the military position" by others. Its obstetric dignity is well shown by the statistics of Ahlfeld who found 38 cases in 3000 labors (1.26 per cent.). This percentage is only a trifle less than the total sum of occipito-posterior positions which reach 1.5 per cent. of all vertex cases. Torggler, of Prague, in 1888, reported 34 cases which represented a frequency of 1.5 per cent. in his service.

In the service of the Chicago Lying-in Hospital, it is the practice to make examinations as infrequently as possible, hence a certain number of so-called "slow" or "tedious" labors might be cases of deep transverse arrest which escaped notice, through spontaneous rotation after some delay. Nevertheless, a review of the records has shown that in a series of 3600 labors, the position was recognized and required treatment 34 times, or 0.97 per cent. of the cases.

It has been repeatedly observed that the difficulty occurs most frequently with the first child; thus of Ahlfeld's 38 cases, 27 were primiparæ. In Torggler's series the position was observed in primiparæ five times as often as in multiparæ. Münch says that 81.61 per cent. of the cases occur in primiparæ. These statistics are not supported by the Chicago Lying-in Hospital reports which show that in 19 cases the women had borne from two to eight children, and only 15 occurred in the first labors. The intention is of course to show that primiparity is an etiologic factor in the occurrence of the position, and in this opinion, the weight of evidence seems to lie.

Münch believes that the preponderance of cases in this class is due to the early descent of the head into the pelvis during the last three or four weeks of pregnancy, and that during this period it remains in a more or less transverse and slightly deflexed condition until the onset of labor. When labor begins, the head has reached a point where the factors which guide the occiput forward work at a disadvantage, the slight degree of deflexion being sufficient not only to disturb the mechanism of rotation, but a longer diameter (the suboccipito-frontal) must now be acted upon by the expelling forces. The head has only a short advance to make

before reaching the pelvic floor, and as rotation must be accompanied by some advance of the head, the most that can be secured is the rotation of the occiput into one of the oblique diameters, where the labor stops.

It is probable that in primiparæ the soft parts grasp the head much more closely, and yield less readily to the powers of rotation, while coincidently the powers of rotation, naturally undeveloped, are more quickly exhausted, so that both the powers and the resistances combine to prevent the forward rotation of the occiput.

In the cases at the Chicago Lying-in Hospital there are present impoverished conditions and unsanitary surroundings which favor defective innervation, weak pains, contracted pelvis, and other factors of considerable etiologic importance. It is possible that these women have attained the acme of their physical development at the time of marriage, and thereafter excessive work, in addition to the care and nursing of children, so weaken the nervous and muscular systems that the danger of weak pains, *inertia uteri,* and insufficient action of the abdominal muscles, increases with each pregnancy, and the importance of any other etiologic factor becomes greatly intensified.

In flat pelves of high degree, the head does not enter at all without interference; but in flat pelves of moderate degree the head frequently engages in the transverse diameter with the large fontanelle on the same or a lower level, than the small, because of the shorter bi-temporal diameter, and anterior rotation is delayed until the head reaches the small pelvis, where the proportionately small saggital diameter together with the deflexion of the fetal head prevents rotation, and the head reaches the pelvic floor in the transverse and maintains that position.

The generally contracted pelvis also is responsible for a certain proportion of the cases (10.5 per cent.). Zweifel reports a case in which an over-development of the frontal bone caused it to become incarcerated in the obturator foramen, and rendered further rotation impossible. Other conditions may arise, such as the prolapse of an arm between the head and the anterior pelvic wall, which mechanically prevent rotation.

It is very interesting to note that 47 per cent. of Ahlfeld's cases occurred in pelves that were too large, either actually or relatively

and I have met with several cases of this character which seem directly to contradict the statements made when discussing primiparity as an etiologic factor.

Let us consider for a moment the active agencies in the rotation forward of the occiput: First we have the "law of the pelvic floor," which may be briefly stated thus: "Whatever portion of the fetal head first strikes the pelvic floor is rotated forward." Secondly, we have the "law of accommodation" of Pajot, which is: "When a solid body is contained within another and either body or container is subject to alternations of activity and repose, the body tends without ceasing to conform to the shape of the container."

Now in normal cases the occiput first strikes the pelvic floor and is rotated to the front. In normal cases the head in a state of extreme flexion will attempt under the activities of the pelvic floor, and the *vis a tergo,* to accommodate its diameters to the most suitable diameter of the outlet, namely with the occiput to the front. In the "too large" pelvis the head finds plenty of room in any position and is not compelled to hunt for space, while on account of the relative disproportion between the head and the pelvis, the head does not fit the grooves, the mechanism of the act is disarranged, and the pelvic floor loses its power to drive the occiput forward. In fact, if it acts at all, the entire head must go forward since the entire vertex strikes the pelvic floor at the same time, and the head may be forced to the pelvic outlet in the transverse and there remain. In the too large pelvis, the bag of waters also may suddenly rupture under the stress of powerful pains which then force the head into the pelvis in a transverse position where it becomes arrested. This case will correct itself if pains are strong, or the pelvis large enough, by the delivery of the head unrotated, as we shall see later.

Only a small hindrance is necessary to prevent rotation when the mechanism becomes imperfect; thus ankylosis of the coccyx, a protruding sacrum, or exostoses of the symphysis or sacrum may easily produce this result.

In another class of cases, the head and the pelvis may be normal, and the powers of labor so weak that the "turning mechanism" does not functionate and the occiput does not rotate. The head may

descend with the occiput anterior or posterior, and rotation may occur into the transverse where the process stops.

By disturbing the flexion of the head, and by favoring engagement in anomalous positions, a pendulous abdomen may also be regarded as causative. When the fetal head is abnormally long antero-posteriorly (dolicocephalus) the danger of impaction is greater, because the two arms of the lever are nearly equal in length, and flexion does not occur. Whatever the cause, the necessity is apparent of knowing accurately the position of the head.

The diagnosis is readily made by vaginal examination, the head is found on the perineum or beyond the midplane, the saggital suture is transverse, the fontanelles palpable at about the same level on opposite sides of the pelvis, and the progress of labor is arrested (Fig.1).

This arrest of the labor entails great danger for the mother and the child. The pains may cease and to the mother accrue all the evils incident to a prolonged labor, including exhaustion, and possibly sepsis, or ruptured uterus. Here too will be found necrosis of maternal tissues from pressure, with vesico-vaginal or recto-vaginal fistulas. The child may become infected, the scalp or cranium necrosed, or the cord compressed. The child may die from traumatism, or as a result of the prolonged labor.

It is true that these cases frequently terminate favorably if given time enough, but the condition of the child or of the mother may necessitate early intervention. The spontaneous solution of the problem may occur in several ways: The occiput may rotate anteriorly and the case terminate normally, or it may rotate into the hollow of the sacrum and remain as an occipito-posterior position, or greater deflexion may occur, the large fontanelle sinking lower than the small one and taking the line of direction produces a forehead presentation; or if the pubic arch is large enough, the vagina and perineum distensible, and the impelling force strong, the head may be delivered in the transverse position, as in four of Ahlfeld's cases, one of Torggler's, and one of my own.

The treatment varies with the conditions in each case, but an incorrect interference can be productive of more harm to the mother or the child than if the case were left to nature.

When no necessity for immediate delivery exists, the woman

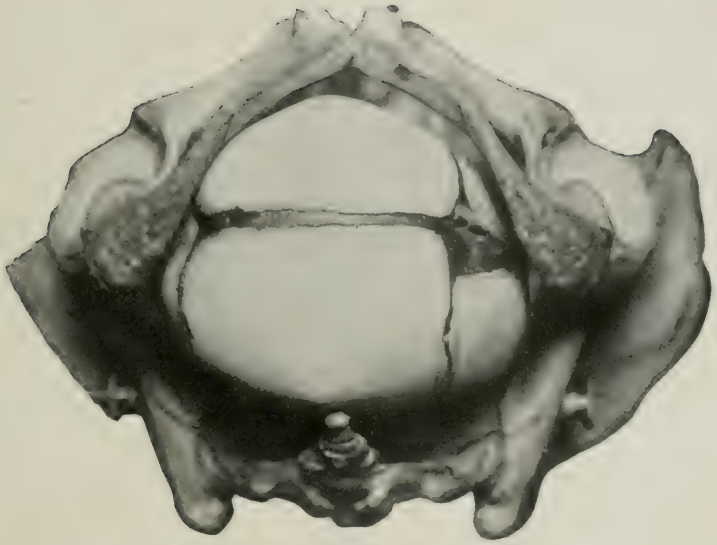

FIG. 1.—The fetal head in the right occipito-transverse position.

for an hour or so should be placed upon the side toward which the occiput points; this permits the occiput to sink deeper into the pelvis, flexion is facilitated, a smaller diameter (suboccipito-bregmatic) is brought into relation with the plane of the pelvic strait, and in consequence rotation frequently occurs, and the case terminates spontaneously.

If this fails, two fingers (Tarnier) or the half hand may be carried in behind the posterior parietal bone and an attempt made to assist the forward rotation, but it is more probable that this manœuvre will succeed in cases due to large pelvis or small head. In case of failure by the preceding methods or before they are tried, pressure may be made upon the sinciput upward during a pain to secure flexion (Hodge). The *vis a tergo* may be increased in selected cases by the Kristeller expression.

The average duration of the second stage in Torggler's series was two hours, and in the Chicago Lying-in Hospital series about four hours, as the forceps are only applied in this service under very strict indications. In danger threatening the mother or the child, the labor should be immediately terminated with the forceps, but otherwise interference should be delayed until it is evident that the head will not rotate spontaneously. If manual correction fails, or if no progress occurs within two and a half hours after complete dilation of the os, the forceps may be applied, and indeed, in such cases the forceps are indicated to complete the delivery. It is nearly impossible for a case of this kind in a primipara to terminate spontaneously and most will be delivered by forceps.

From a study of cases it is probable that of all instances in which forceps are applied under strict indications, the head is found in deep transverse arrest in approximately 35 per cent. Twenty-four of Ahlfeld's cases were terminated with the forceps, danger to child constituting the most frequent indication for forceps in this series.

In the Chicago Lying-in Hospital service, forceps were applied twenty-four times, and in ten cases rotation occurred without artificial assistance. In this series, maternal conditions most frequently furnished the indication for interference. A slight traction will many times start the rotation and the labor easily terminates.

It was in this class of cases that the vectis formerly found its greatest utility. In the peculiar application of the forceps lies the

necessity for accurate diagnosis. The forceps blades are constructed for application to the sides of the head, but if so applied in " deep transverse arrest " they will lie in the antero-posterior diameter of the pelvis which is not possible for forceps with a pronounced pelvic curve; neither can the blades lie in the sides of the pelvis, because the fetal head would be grasped in the antero-posterior diameter which is a bad position for the head and an unfavorable one for the forceps. Hence a compromise is necessary and the blades must be applied in one of the oblique diameters with the pelvic curve toward the occiput. If the occiput lies to the woman's left, the forceps should be applied in the left oblique (that is, a line passing between the blades lies in the left oblique diameter of the pelvis), the right blade resting on the anterior malar bone and the left on the posterior parietal protuberance. When the occiput points to the right, the forceps lie in the right oblique, etc.

The application presents some difficulties. The patient being in the dorsal position on the table with elevated hips, the left blade is passed along the palmer surface of the right hand into the hollow of the sacrum behind the head and allowed to remain there if the occiput points to the left, while the right blade is introduced into the hollow of the sacrum and carefully rotated under the guidance of the fingers, over the face of the child to the anterior malar bone. (Fig. 2).

If the occiput lies to the right, the left blade is rotated into position over the anterior malar bone before the right blade is introduced.

Since the head is grasped in a large diameter, care must be used to avoid too much compression on the head during traction, hence a finger or towel should be placed between the handles.

Two objects must be kept in view—rotation and extraction. The occiput should not be rotated forward directly as some advise, for fear of lacerating the vaginal walls, but traction and rotation must be simultaneous, and about in the proportion of two of traction to one of rotation. Occasionally, however, the vagina will be torn in difficult cases, in spite of every precaution.

After each traction the handles should be relaxed and the sutures and fontanelles examined to note progress. Readjustment of the blades should be made as often as necessary until they lie in

FIG. 2.—The forceps applied in a case of right occipito-transverse position—the left blade to the posterior parietal bone, the right blade over the anterior malar bone. Note the separation of the handles of the forceps.

the sides of the pelvis and grasp the sides of the fetal head. After rotation occurs the blades *may* be removed and if the pains are strong, nature may be allowed to terminate the case, assisted possibly by Ritgen's manœuvre.

Usually the rotation will be followed by immediate extraction, since the necessity that requires interference carries with it the demand for delivery.

The use of the axis traction instrument for all cases requiring forceps is the rule in Scotland and Dublin, and the practice is not uncommon in France. In this case the axis traction forceps may be applied directly to the sides of the pelvis, irrespective of the position of the head; rotation then occurs without interruption from the blades, and the results are generally satisfactory.

If the child is living and forceps have failed, symphyseotomy may be considered. If the child is dead, no attempt should be made to apply forceps; but in behalf of the mother, the indication is absolute to perforate, crush, and finally extract the diminished head.

To Summarize: (1) Deep transverse arrest of the head is a relatively common complication of labor.

(2) The diagnosis is easily made from the position of the sagittal suture, and the fontanelles.

(3) The normal termination of the case cannot be waited for in most instances, but forceps should be applied as soon as it is evident that rotation will not occur spontaneously.

(4) In the absence of danger to mother or child, two or three hours may elapse without interference.

(5) Location of the occiput must be determined before the blades are applied.

(6) The blades should be applied in that pelvic oblique diameter toward which the occiput lies.

(7) Traction and rotation must be simultaneous.

(8) When the child is dead, craniotomy is imperative.

THE PELVIS OF LAME WOMEN

BY A. PINARD, M.D.

Professor of Obstetrics at the Paris Faculty

GENTLEMEN: The presence still in our wards of three lame women recently confined here, leads me to speak to you to-day on the question of the pelvis vitiated by lameness, from an obstetrical point of view.

The study of the pelvis of lame women has generally been treated from an *analytical* standpoint, and many are the papers that have appeared on the pelvis of women with hip-disease, with congenital dislocation of the femur, with infantile paralysis, etc. But to-day my intention is to examine this subject from a *synthetic* point of view, to see what the essential characteristics are of the pelvis of lame women, and, placing myself on clinical ground, to endeavor to introduce some order into a question that analytical descriptions of exceptional cases have undoubtedly thrown into confusion.

The obstetrical history of the pelvis of lame women dates from the seventeenth century. You will find in the tenth chapter of Peu's work, given up to the confinement of lame women and hunchbacks, an account of the troubles to which they are exposed, particularly during pregnancy, and the relation of a personal case.

"I remember," he says, "that at the time of the second Paris war, when I had just begun practice, it was suggested that I should wed a young woman, beautiful, rich and very intelligent, the daughter of a man for whom I had a great respect, but who was small and very lame on one side. The results which I apprehended in these circumstances decided me not to entertain the proposal."

De la Motte is less severe. In the chapter of his treatise that describes the confinement of lame women and hunchbacks, he says: "M. Peu wrote such a tirade against women afflicted with one or the other of these infirmities, that anyone reading his book would conclude that all idea of marrying ought to be absolutely forbidden in their case. Although the young lady that he might have married,

and that another man did marry, was lame, and might have had a very bad confinement, is that a sufficient reason for inferring that all lame women are exposed to such a misfortune? It is to be feared that a feeling of spite led this writer to utter this anathema against all women suffering from this infirmity, so that they would feel that they could not possibly escape from this wretched predicament."

And De la Motte adds: "This would result in the most unfortunate consequences for them, who are as anxious as others to marry; whereas we often see perfectly-made women giving themselves up to the Lord, and burying themselves alive, so to speak, in the silence of the cloister."

Furthermore, history contains the case of a celebrated lame woman, a favorite of the great king, Mlle. de la Vallière, whose confinement gave rise to no difficulty, in spite of Mauriceau's aphorism: "Misshapen and lame women have much more troublesome confinements than other women."

In connection with Mlle. de la Vallière, it has been said that her confinement was the occasion for the first appearance of surgeons in assisting women in labor. Thus Sue, in his historical essays, writes: "M. Astrue says that he was assured that the date when surgeons were first called on in confinements, does not go back beyond the first labor of Madame la Duchesse de la Vallière in 1663 . . . . which set the fashion among the princesses of having surgeons attend them on such occasions. This habit soon became general, and gave rise to the new term of *accoucheur.*"

Thus, according to Astruc, the first accoucheur was Julien Clément. This is a mistake. The accoucheur of Mlle. de la Vallière was Francois Bouchet, and Francois Bouchet was not the first surgeon to be called in in a case of labor. In proof of this I have only to recall to your memory the names of the celebrated French accoucheurs of the sixteenth and seventeenth centuries: Ambroise Paré, Franco, Guillemeau, Mauriceau, etc. But let us return to the lame women.

With Levret (1761) the elements of the prognosis of confinements in the case of lame women, still considered as a general class, become more exact: "Lame women who, *during their childhood, had no softening of the bones,* bear children as a rule as easily

as those who are not deformed or lame, when the deformity did not take place until after the age of puberty; but it is rare that such should be the case when lameness occurred during the early years of life."

During the last century, among the many publications concerning pelvic malformations, an important place was given to the pelvis deformed by lameness,—either by congenital dislocation, by hip-disease, or by infantile paralysis. Thus the theses of Guéniot, 1869, Tracon, 1889, and Prourost, 1891, were devoted to the analytical study of these cases.

I shall content myself with these short remarks about the historical side of the question, in order to lose no time in getting to the synthetic study of the pelvis in lameness.

Any woman who can only throw her weight partially on one leg is lame.

Have all lame women a vitiated pelvis? No.

Only women who go lame before the complete development of the pelvis, that is to say, before about the sixteenth year, have their pelvis deformed.

These deformities, produced by lameness, appear with characteristics that are always the same. Whatever may have been the cause of the lameness, whatever may be the seat of the causative lesion,—congenital dislocation, hip-disease, knee arthritis, clubfoot, amputation of the thigh or leg, arrested development of the leg through infantile paralysis, etc., and at whatever age the lameness may have begun, the deformity is always the same.

You will see it plainly on these specimens of the pelves of children having succumbed to coxalgia, for which I am indebted to Dr. Ménard, of Berck-sur-mer.

Set aside everything that is outside of the pelvis, and concentrate your attention on the pelvic cavity.

What do you see there?

On the healthy side the linea innominata of the iliac bone is straightened out, flattened.

On the diseased side, the iliac bone is developed to a greater or lesser extent; in many cases it is atrophied; but the general form and the curve of the linea innominata are respected.

The sacrum is asymmetric; its wing on the healthy side is less

developed than on the diseased side. This lack of symmetry, of the healthy wing of the sacrum, to which I have for a long time been calling attention, has been carefully studied under my supervision by Dr. Raoux.

These are the essential features, the regular characteristics of the pelvis vitiated by unilateral lameness.

They result in unequal transmission of the weight of the body, to the lower limbs. On the healthy side the predominating femoral counter-pressure tends to straighten out the iliac bone and hinders the development of the wing of the sacrum.

But, although the pelvis is deformed, and although the relative dimensions of its useful diameters differ from the dimensions looked on as normal,—it remains, in its entirety, sufficiently roomy, to admit of spontaneous delivery at term.

Is this to deny that the cause that produces the lameness does not by itself sometimes vitiate the pelvis, and does not add to the essential deformities I have just described, deformities of another kind? Certainly not.

It may happen that during the evolution of a tuberculous arthritis of the hip-joint, the lesions affect not only the outside of the pelvis, but the inside also; in this way are produced bony deposits, intra-pelvic abscesses, and intra-pelvic dislocation of the femoral head by destruction of the surface of the acetabulum. In such cases the pelvis is not only deformed by the lameness but by the local lesions, and the vitiation is a complex one. But you must always remember that such eventualities are entirely exceptional; in the interesting publications to which I referred above, you will find an account of these unusual cases published owing to their rarity.

There is another cause of lameness that is sometimes accompanied by very pronounced lesions of the pelvic frame. In infantile paralysis the check in development may concern the iliac bone, and reduce its exterior dimensions very considerably.

We once had in this clinic a case of this nature, but in spite of the enormous atrophy of the iliac bone and lower limb, the latter reduced to a stump without shape appended to the body, spontaneous delivery occurred without hindrance. This is accounted for by the fact that whatever may be the atrophy of the iliac wings and of the

ischio-pubic branches, the amplitude of the linea innominata remains sufficient.

Thus in coxalgia the causal lesion may make the deformity worse; in infantile paralysis it does not do so.

Finally, to the deformities that I described above as the consequence of simple lameness may be added the deformities produced by rachitis. The coexistence of these two factors of pelvic deformity, lameness and rachitis, produces a type of complex vitiation that gives the greatest trouble in labor. For to the lack of symmetry of the pelvis is added the lessening of the antero-posterior, sacro-pubic diameters.

Such are the general ideas on this subject that I ask you to remember, and that can be summed up as follows: The pelvis of women who have been lame before the age of sixteen is deformed, but this deformity has no obstetrical importance unless an additional lesion, and specially rachitis, gives rise to a complex vitiation of the pelvis.

I now come to the clinical application of these general ideas.

We have, as I told you, three women in our wards lame from childhood but whose confinement has been normal. One of them has been lame since the age of two and a half through infantile paralysis. The other two have been lame through tuberculous arthritis of the knee, the one since the age of three, and the other four. Here is a short account of their obstetrical history.

Case I.—A woman of 33 years, said to have been born at full term; was nursed by her mother, showed no sign of rachitis, and began to walk at the age of eighteen months. She was seized with infantile paralysis at two and a half years. The paralysis was localized in the lower limbs and especially the left side.

After an interruption of four months she began to walk once more with the help of orthopedic apparatus, which she continued to wear until the age of nine; from that time on, she used nothing but an orthopedic shoe on the left side.

Present Condition.—Left foot in varus equinus; lower limb on that side markedly atrophied, and shorter than its fellow (four centimeters difference in the distance between the iliac spine and the point of the external malleolus). On the diseased side there are cicatrices of chillblains on the toes, some hard edema on the lower

portion of the leg, and a slight degree of thickening of the tibia. Lumbar scoliosis with convexity to the right. No signs of rachitis.

History of her three confinements:

(1) 1898. Spontaneous confinement at the Maternité; apex presentation; child weighed less than 2,000 grams, and died on the twentieth day.

(2) 1899. Spontaneous confinement at nine months at Baudelocque Clinic; apex presentation; child weighing 2,820 grams, with a bi-parietal diameter of 9. cm.; still living.

(3) 1905. Spontaneous confinement at term at Baudelocque Clinic; apex, in left position, anterior variety; duration of labor, 8 hours 30 minutes; of expulsion, 5 minutes. Living child weighing 3,600 grams, and having a bi-parietal diameter of 9.8 cm.

Case II.—A woman of 22 years. Walked at the age of fourteen months, and never showed any signs of rachitis.

Was treated at Trousseau Hospital at the age of two for pulmonary congestion. A few months after leaving the hospital appeared the first symptoms of a disorder of the left knee which was diagnosticated as a "white swelling" by the physicians at the Trousseau Hospital.

Treated by means of plaster bandages; the child continued to walk during almost the entire duration of the complaint; she was kept in bed only a few weeks on the occasion of an attack of measles with complications.

The articular disorder disappeared about the age of four after the elimination of a sequestrum. In spite of this recovery, tendinous retraction occurred, throwing the knee into a defective position of permanent flexion that became more and more pronounced, and for which the joint was resected at the age of eleven years.

Actual Condition.—Lower left limb somewhat atrophied and slightly shorter than its fellow (2 cm. difference in the lengths taken from the iliac spine to the external malleolus); the knee presents limited movements of flexion, and abnormal mobility in the lateral direction. Lameness only slight. No signs of rachitis.

Confinement at Baudelocque Clinic in 1905. Premature rupture of the membranes three days previous to the confinement, which led the patient to decide to enter our wards. When she arrived: apex presentation in left position, anterior variety; preg-

nancy in the ninth month. Duration of labor, 3 hours 30 minutes; of expulsion, 30 minutes. Child weighing 3,960 grams, with a bi-parietal diameter of 9 cm.

Case III—*Tuberculous Arthritis of the Knee, and Fracture of the Femur.*—Woman of 35 years; did not begin to walk until she was three years old.

Was taken care of during all of her early youth for arthritis of the right knee, looked on as a "white swelling" that began at the age of four, and was treated by means of plaster and silicate bandages, thanks to which the child continued to walk.

At the age of seven, fracture of the right femur in the upper third, which prevented her walking for only six weeks.

At the age of twenty-one, as the knee was taking a more and more pronounced permanent position of flexion, resection of the joint.

Present Condition.—Right lower limb atrophied, and 8 cm. shorter than its fellow. Straight ankylosis of the joint. Operative scars about the knee, and voluminous callus of the femur. Curvature of the left tibia; flattening of the right hip. In the pelvis the angle cannot be reached.

History of her six confinements, all at Baudelocque Clinic:

(1) 1892. Application of forceps after 60 hours of labor, as the child was in danger. Child weighed 3,830 grams, with a bi-parietal diameter of 9.3 cm.; it died at the age of two and a half of broncho-pneumonia (alcoholic father). Placenta, 810 grams.

(2) 1896. Version by external manipulation during pregnancy. Confinement at the ninth month in apex presentation. Duration of labor, 5 hours. Child weighed 3,760 grams, with a bi-parietal diameter of 9.7 cm. Died at the age of nine from meningitis (father different from the one of first pregnancy, but also alcoholic). Placenta, 460 grams.

(3) 1897. Fetus did not descend into the pelvis during pregnancy. Spontaneous confinement at term. Apex. Child weighed 4,000 grams, living, with a bi-parietal diameter of 9.4 cm. Placenta, 660 grams.

(4) 1899. Breech presentation and version by external manipulation during pregnancy. Confinement at nine months, apex,

spontaneous delivery. Child weighed 3,920 grams, with a bi-parietal diameter of 9.2 cm.; still living. Placenta, 800 grams.

(5) 1901. No accommodation during pregnancy; presentation first of one side, and then of the breech; version by external manipulation; spontaneous confinement at nine months, apex. Child weighed 3,930 grams, with a bi-parietal diameter of 10 cm. Brought up at a *créche;* died at ten months, of measles. Placenta, 600 grams.

(6) 1905. Spontaneous confinement at nine months; presentation of the apex; living child weighing 3,380 grams; bi-parietal diameter 9 cm. Placenta, 480 grams (The father was a different man from those of the foregoing confinements).

These three lame patients, whose obstetrical history has been a fortunate one, were not women who had had coxalgia, and I am aware that of all the pelves of lame women it is the pelvis of the coxalgic that is held up to you as the least favorable.

But, let us turn to the facts in the case, and look through our records, as was done by Bruneau (1892) and by Ortel (1904).

In 1892 Bruneau wrote his thesis on the cases of women with hip-disease who had come to my wards at Lariboisière and Baudelocque to be confined. He adopted as its title the phrase: On spontaneous confinement in cases of coxalgic pelvis. "In most instances," he says, "when the lesion is a simple one, the confinement is not impeded in any way." This conclusion did not agree very well with the opinion generally held on the subject.

The cases that we have observed at Baudelocque Clinic since then have confirmed these conclusions of 1892. In round figures we have had about 80 per cent. of spontaneous confinements. When there has been trouble it has always been with pelves that had some complex vitiation.

In practice, then, your duty will be to detect these complex vitiations. You must look for:((1) Rachitis; (2) intra-pelvic tubercular lesions. When your examination does not reveal one or the other of these factors, you can make a favorable prognosis.

But when you detect them, be guarded in your forecast, as you may find yourself in the presence of a most serious case requiring Cesarian section. But fortunately, I say it once more, such cases are the exception.

You can therefore forget Peu's pessimistic advice to such women: "Remain in bed, be sober in all things, and take no violent exercise; when this is not sufficient, *do not marry.*" Forget also Siebold's commentary on this final remark: "which is undoubtedly the best advice that could be given to them."

If you are consulted as to the advisability of marriage in such a case, reply: Yes. If you are questioned as to whether such a married woman can have children, examine and see whether the lameness has been the only cause of the deformed pelvis. When you find yourself in the presence of a pelvis with complex vitiation, —an exceptional case,—you must be on your guard. Otherwise you are warranted, and such are the usual circumstances, in reassuring your patient as to the favorable issue of any future pregnancy.

Rhinology

CLINICAL RHINOMETRY: FUNCTIONAL EXAMINATION OF THE NOSE

BY MARCEL LERMOYEZ, M.D.
Physician to the Paris Hospitals

THE time is at last gradually drawing near when every physician with a proper respect for his calling will know how to examine the nasal cavities. In the digestive tract our inquisitiveness has led us everywhere, from visual inspection of the tongue to digital examination of the rectum: every portion has been investigated by workers with illustrious names. The respiratory system, on the other hand, less easy of access, appears to confine us within more moderate bounds; as a matter of fact, nearly all physicians, and possibly even those in particular making a specialty of lung disorders, limit their examination to the lower portion of this tract. I am at a loss to account for this difference, and do not quite see how anyone can; for, after all, is it less difficult to auscultate the lungs than to examine the nose with a speculum? For the nose is far from being a negligible factor in respiratory pathology!

We are forced to admit, then, that this state of affairs is not in accordance with good sense; let us therefore train ourselves to examine the respiratory tract from its beginning to its end, by means of the speculum as well as the stethocope.

Even so, the practitioner will make a mistake if he limits his nasal ambition to the handling of the speculum. He ought to, and can, do better than that, as I shall attempt to show him in the following paragraphs.

An examination of the eye is incomplete if it is confined to a simple objective exploration of that organ without an estimate of its functional value; thus ophthalmoscopy has to be completed by ophthalmometry. In the same way, rhinoscopic exploration of the nasal fossæ does not suffice to make a diagnosis; to it must be added the functional examination of the nose. Now it happens, for the

satisfaction of the general practitioner, that the latter exploration is the easier of the two, and at the same time gives more useful information from the point of view of clinical medicine.

The nose serves two purposes: to breathe and to smell; a breathing organ, leading the air to the lungs, is combined with a sensorial organ, allowing odors to reach the olfactory nerve. These two functions have been happily assigned by nature to a single organ, since they lend each other mutual help. Perception of smells cannot be acquired save through inspiring air; and in exchange for services rendered, this perception keeps the respiratory tract informed as to the quality of the air it receives.

Still, these two functions are, none the less, quite distinct, and their value has to be measured separately. Thus a functional examination of the nasal fossæ comprises two tests:

(1) A functional test of the nose as a breathing organ,—or Rhinometry.

(2) A functional test of the nose as a special sense,—or Olfactometry.

In the present paper the former alone of these two tests will be considered, the other being reserved for a future occasion.

Rhinometry,—an imperfect though convenient term,—purposes to estimate the degree of permeability of the nasal fossæ to air. It measures their respiratory field.

The Breathing Function of the Nasal Fossæ.—It is the duty of the nose to prepare the air on its way to the lungs, just as the mouth subjects to a preliminary modification the food destined for the stomach.

The nose warms the inspired air. This heating-process (E) takes place according to Block's formula: $E = 5\ (\frac{T \cdot t}{9})$, in which T represents the temperature of the body, and t that of the surrounding atmosphere.

The nose charges the inspired air with aqueous vapor. When the air reaches the entrance to the larynx, it is already almost saturated with moisture.

The nose sterilizes the air we breathe. It stops mechanically the dust, which lodges in its numerous folds, whence it is again expelled by sneezing and blowing. It also destroys, by a special bactericidal action, the germs that reach it.

The mouth is also able, it is true, to conduct air to the lungs; but as it was not intended for this purpose it fills it very imperfectly. Its thick, smooth mucous membrane can in nowise replace the pituitary surface, of which it possesses neither the vascularization nor the abundant folds. The consequence is that it is not at all a matter of indifference to the lower air passages whether the air reaches them via the mouth or via the nose; when the nose is inadequate the mouth can only take its place imperfectly. This is why nasal insufficiency is such an important factor in creating disease, a factor which rhinometry enables the physician to estimate with perfect ease.

Path Followed Through the Nose by Inspired Air.—It is manifest *a priori* that, in order that the olfactory and respiratory functions of the nose may be regularly performed, the inspired air should follow in it a fixed direction, bring it into contact with the portions of the pituitary membrane destined for those purposes. Paulsen has shown that the direction followed through the nose by the air is invariable under normal conditions. The nose, through its interior conformation, obliges the column of air that enters to follow a certain path, directing it upward toward the olfactory region, while at the same time the obliquity of the nostrils inclines the air-stream toward the septum, along which it slips without running into the hollows of the external portion of the nasal fossæ.

The inspired air describes an arc whose convexity reaches the edge of the upper turbinate, without penetrating into the olfactory passage. The portion of air entering through the anterior half of the nostril occupies in the nasal fossæ the highest position, and is alone used for smelling; the portion entering by the posterior half of the nostril goes less high and follows a shorter path toward the naso-pharnyx: its use is purely respiratory.

The direction followed on the way back by the expired air is exactly the reverse of this.

What might be referred to as the three guide-posts along this route through the nose are: (1) Kiesselbach's zone, on the anterior portion of the septum; (2) the head of the middle turbinate; and (3) the tail of the lower turbinate. At the first two of these points layers of dust can often be found, showing how the air infringes on

them as it enters. The least abnormal enlargement at any one of these three points gives rise to a mischievous obstruction.

It may seem surprising that the current of air reaches neither the olfactory region, the seat of the sense of smell, nor the middle portion of the lower turbinate, whose erectile tissue is so suited to warm it. But there is, of course, no point in the nose where the air is absolutely motionless; in reality the inspired air does reach the zones referred to, but only by diffusion, out of regard for the delicacy of the olfactory cells.

Necessity of a Rhinometric Technic.—The permeability of the nasal fossæ cannot be determined objectively nor subjectively; rhinometry alone enables us to estimate it.

Objectively we must accord only very limited confidence to what we ascertain by rhinoscopy. Obstruction and nasal insufficiency are, contrary to what is generally held, two non-synonymous terms, whose variations do not follow a parallel evolution.

Thus, a given nose may seem badly obstructed on the right side by a spur of the septum, and yet rhinometry will show that breathing is insufficient on account of an enlargement at the posterior end of the left turbinate.

Again, we may be tempted to consider as sufficiently permeable a nose whose lower turbinates are retracted enough to enable us to see the movements of the palate, whereas we are neglecting the importance of a hypertrophy of the head of the middle turbinate which is barring the road to the inspired air. I have published cases of absolute functional insufficiency of the nose, in which the cavities were anatomically permeable in appearance; in these instances rhinoscopy failed entirely.

Subjectively, without rhinometry there is every chance of making greater errors still, through relying on the patient's sensations.

One person, whose nose may be altogether insufficient, may not even suspect the existence of this condition because the nasal stenosis was acquired gradually, because his system long ago grew accustomed to this unsatisfactory state of affairs, and because the limited nasal field suffices for the slight exertions that the patient may have to make in ordinary life; the nasal inadequacy is only revealed on some occasion when the lungs are called on for some real effort.

This is the case with the majority of patients who come to us for throat trouble, and who are amazed that we should discover nasal stenosis.

On the other hand, another patient, with tabes, or ozena, may have a nose sufficiently permeable, too much so, perhaps; and yet he will fancy that he breathes with difficulty because the dulled nasal faculty of sensation keeps him only partially informed as to the volume of air passing through the nasal fossæ.

Principles of Rhinometry.—Rhinometry may be: (*a*) qualitative; (*b*) quantitative.

Qualitative Rhinometry—This is a simple but very imperfect method, which for a long time was all that physicians demanded.

Various rapid means enable one to ascertain very approximately the greater or lesser degree of permeability of the nose. For instance: close one nostril with your finger, and make ample inspirations and expirations through the other only. The noise thus produced varies according to cases: when the nose is spacious the sound is low, something like an F; when narrow it is more acute, like CH. Or, place the back of the hand a few centimetres below the nostrils and estimate the strength of the expired current of air for each one of them by the impression received on the skin. Again, ask the patient to inspire vigorously, and watch to what degree the nostrils close in; the freer the nasal cavity, and the more copious the inflow of air, the more they will be drawn toward the middle line.

It should, however, be noted that such breathing, always somewhat forced, easily surmounts slight obstacles which, under normal conditions, oblige the patient to keep his mouth unconsciously open.

Quantitative Rhinometry.—This method, more complex but more exact, endeavors to measure the degree of permeability of the nasal fossæ. This test can be made in two ways: directly, or indirectly.

Direct Rhinometry.—The classical method of measuring the volume of air passing through the nose consists in getting the patient to make expirations alternately through each nostril into a spirometer, or, better still, simultaneously through both nostrils into two different spirometers. Mendel constructed a very exact apparatus on this principle, and made a very interesting comparison between

buccal and nasal breathing; but his is a laboratory apparatus rather than a clinical instrument.

Another method consists in measuring the time required by a given amount of air to pass through the nasal fossæ. If the pressure remains the same the duration of the passage is in direct proportion to the degree of nasal stenosis. Kayser imagined an ingenious apparatus for rhinometry on these lines; but here again, its use is too complicated for current purposes.

These methods of direct rhinometry, therefore, are not suited for the practitioner, who requires, above everything, a rapid means of testing this function. Fortunately, Zwaardemaker has supplied a much simpler solution to this rhinometric problem, in the following way:

Indirect Rhinometry.—Expired air is saturated with aqueous vapor; at 37° C. it contains 42 milligrams per cubic meter, and this saturation is constant. If, therefore, we can estimate the amount of aqueous vapor escaping from the nostrils, we can thereby ascertain indirectly the amount of air that has issued from them. In order to acquire approximately this unknown quantity, we have only to place at one centimeter below the nose some cold and polished surface; the expired aqueous vapor will then condense on it in spots of moisture, called respiratory spots. These spots indicate, if not in a very precise manner, at least approximately, the degree of nasal permeability. They are oval in shape. In some cases, during their evaporation, they separate into two portions, divided by an oblique band, which, in Zwaardemaker's opinion, corresponds to the relief of the lower turbinate. The antero-external spot would then be attributable to the column of air passing above this turbinate, and would give a fair measure of the olfactory field.

Technic of Rhinometry.—(*A*) Glazel's mirror is the simplest instrument for performing rhinometry quickly. It gives instant and quite sufficient information concerning nasal permeability.

This mirror is made of a smooth plate of nickeled metal carefully polished, on which spots of vapor are much more apparent than on a sheet of glass; to its sides are fitted handles for the operator's convenience; at its ends are notches that adapt themselves to the patient's upper lip. Both surfaces are laid out in arcs of circles, by which means the extent of the vapor spots can be measured.

The use of this rhinometer is really very easy. The patient should be asked first to blow his nose, to free it of any secretion that might act as an obstruction; he should then breathe quietly and regularly in his normal manner. When the respiratory rhythm is well established, the mirror should be placed horizontally half way between the nostril and the upper lip, and should be left in place during two or three expirations. The mirror should then be rapidly turned, and its other extremity placed below the patient's nose. In this way two pairs of respiratory spots can be obtained in a few moments, one fresher than the other, and consequently, at successive phases of evaporation. These spots take about a minute to disappear, and while doing this they grow gradually smaller, and thus accentuate their differences.

There are two ways of reading these respiratory spots:

(*a*) Their length of evaporation can be estimated, which is in direct proportion to the amount of vapor, and consequently, to the quantity of air expired. (*b*) The area they cover can be estimated. In normal conditions two symmetrical spots like a butterfly's wings should be observed, extending to the fourth arc of a circle. When a nose is inadequate on one side, there may be no spot at all on that half of the plate; but, as a rule, a certain area is moist, though it does not extend very far.

(*B*) De Courtade's pneumodograph. The technic of this instrument is somewhat more complicated, but it has the advantage of indicating, together with the degree of nasal permeability, the amount contributed by the mouth in the respiratory act. By its means, therefore, it is easier to estimate the degree of nasal obstruction, from a physiologic point of view.

This apparatus is composed of three supposed plates of glass: the first, vertical, is placed in front of the mouth at a distance that can be varied; the second, horizontal, comes below the nostrils; the third, vertical, and in an antero-posterior direction, divides the preceding one in two equal halves, while its upper edge by being brought into contact with the sub-septum, separates the breathing of the right nostril from that of the left.

Unfortunately the respiratory spots are, with this instrument, very evanescent; for this reason de Courtade has devised an ingenious method of obtaining a permanent record of them.

To do this the apparatus should be placed so that the little antero-posterior plate is brought to bear both on the sub-septum and lip, and the patient is requested to breathe in a natural manner, as he is accustomed to, without thinking of the instrument. After ten or fifteen expirations the instrument is removed, the little antero-posterior plate rapidly taken off, and a piece of paper prepared with aniline or a ferrotannic salt specially for this purpose, is spread over the horizontal plate. When brought into contact with the moisture of the plate, colored spots, pink in the first place and black in the second, are formed. In this way can be obtained a permanent record demonstrating the nasal impermeability, and suitable for illustrating case-histories.

Causes of Error in Rhinometry.—(1) It is desirable that the test should last only a very short time, since when this precaution is neglected the two respiratory spots have a tendency to become equal, and the difference between them is more difficult to detect.

(2) In midsummer the difference between the temperature of the plate (25°–30° C.) and that of expired air (37° C.) is too slight for the aqueous vapor to condense on it. It is necessary then, before each examination, to cool the plate off by putting it in cold water, after which it must be carefully dried. The most favorable temperature is 15° C.

(3) The area of the spots is manifestly dependent on pulmonary capacity, the variation of which in different persons influences the results furnished by the tests. Still, this point has no great importance, since rhinometry deals principally with the relative value of spots, comparing with each other the nasal and buccal impressions. Whatever may be the pulmonary capacity of a patient, the relation between nasal and buccal respiration will be constant with that person.

(4) The chief criticism that has been made of this method is that it measures expiratory inadequacy, but gives no information concerning inspiratory insufficiency. This objection has been entirely refuted by Glatzel. With the exception of some very rare instances of pediculated tumors acting as valves in the nasal fossæ, pure inspiratory insufficiency is usually due to a drawing inward of the alæ nasi, due to atrophy of their cartilage and paralysis of their muscles. Now in such cases the nasal stenosis is so easy to

perceive, even for an ordinary person, that there is really no need of having recourse to the rhinometer.

In other cases the inadequacy is mixed, both inspiratory and expiratory, and since the two subdivisions of breathing are equally affected, it is sufficient that we should be informed about the variations of one of them.

That urethral stricture endangers the well-being of the urinary tract back of it, and that stricture of the nose has a similar effect on the respiratory tract above it,—are two symmetrical truths to which physicians have long attached, no one knows why, very unequal importance, but which we are at last learning to look on with a similar degree of disfavor. But although the treatment of coarcted methods has long been a matter of current practice, the apprehension of nasal stenosis still remains a theoretical matter to a certain degree.

Certainly it is well that a physician should realize on general principles that a lung, like a chimney, can have its draught impeded by nasal obstruction; but it would be much more advantageous for him to have some simple means of ascertaining that in a specific case his patient's lungs are being damaged by a nasal stenosis. To do this he need not send his patient to a costly specialist; all he has to do is to procure one of these cheap instruments, spend a few moments learning to handle it, and then measure for himself. When once he has done this he will not be long in convincing himself of the value of his purchase.

I have heard that in a neighboring country when conscripts come up for medical examination they are required to breathe on this nickel plate, and that in this way the imperial army is freed from the start of men unable to breath properly. Now let me ask: How many physicians are there in France who have seen a rhinometer? How many hospital wards have we that are provided with one? Or even, how many clinics for respiratory diseases are there where they are used? Is our country so well-provided with healthy lungs that we can afford to neglect this means of exploring the respiratory coefficient?

Otology

PRIMARY THROMBOSIS OF THE JUGULAR BULB

BY BARTON H. POTTS, M.D.

Rhinologist and Otologist to the German Hospital, Philadelphia

That the general practitioner is coming to recognize the important part played by aural affections and the possibility of grave intracranial complications arising from these conditions, is shown by the character of the work done by aurists and the results obtained. It is a thing to be grateful for that to-day we rarely hear the advice given that "it is only a running ear, let it alone." The physician realizes that from this focus conditions of the utmost gravity may arise.

But the dangers of the very acute conditions are not yet fully appreciated. The opinion prevails to a considerable extent that the grave complications do not appear until the aural condition has reached some degree of chronicity; that some time is required for the infective process to extend through the mastoid cells and involve the dura, the sinus, or the brain. This is very true if the infection follows this direction. But one very important anatomic fact is not considered; that is, the very close relation of the jugular bulb to the floor of the tympanic cavity. The bone in this region may be as thin as tissue-paper, and sometimes is entirely absent. Given this condition, it can be readily understood how easily infection from an acute suppurative otitis media may extend to the large blood-channel. This is the point not fully realized, the danger of infection from an acute process, and this is particularly true in children. McKernon has reported six cases of primary jugular bulb thrombosis in children.

Every intelligent physician is quick to grasp the significance of a chronic aural history in his patients, especially in those having

unexpected and, except for the aural condition, unexplainable variations in temperature. The formerly classic symptom of fluctuation over the mastoid is fast becoming the exception instead of the rule. In cases in which there is an active inflammation of the mastoid cells the sagging of the upper-posterior part of the membrane and canal-wall is our most important sign, and its persistence our most reliable indication for operation; there are also generally pain and tenderness over this region, but in the class of cases referred to in this paper these symptoms may be absent.

Given a chronic suppurative history with infection of the sinus and there will be usually one or more chills, sweating, possibly vomiting, signs of meningeal irritation, Kernig's sign, possibly a change in the eye ground or pupil, though these last two signs are late or absent entirely. But in infections from acute suppuration all of these symptoms, except the meningeal ones, will probably be absent. What, then, is to be our guide? The answer is, the temperature.

Acute suppuration of the middle ear will always cause a rise in the temperature, even to 105° F., particularly in the young, but upon relief of the condition the temperature falls and remains down. In these acute infections the temperature is characteristic; with, or without, accompanying symptoms there will be a sudden rise of four, five, even eight degrees, and then a gradual drop to near normal or below. The patient seems much improved in all respects and will remain in this condition from a few hours to a day or even longer; then there is again the sudden and extreme rise and this is repeated until operative relief is given. Having made sure by free paracentesis and proper treatment that drainage from the tympanic cavity is unobstructed, these variations of temperature are practically diagnostic. The bacteriologic findings are not conclusive, but they are of importance in giving some indication of the virulence of the infection. The same may be said of the blood count; a polynuclear leukocytic percentage of 80 or over may be taken to indicate infection, but one of my worst cases had a percentage of only 70.

It is doubtless true that there are many cases of acute suppuration in which there is a phlebitis of the bulb or sinus, and even the formation of a clot which speedily breaks down and permits the flow of the blood-current, and nature takes care of the process.

These cases go on to a spontaneous cure, their probable nature being shown by the sudden variations of temperature and later subsidence to normal. The diagnosis cannot be made positively by any one symptom, but by the study of the case and the appearance of the patient; clinical experience alone will be our guide. The location of the thrombus in these cases bears out the teaching of Randall, who has maintained that the acute infection is always at the bulb, while in the cases with a chronic history it is in the sigmoid portion or near the knee of the sinus. Whether the infection occurs directly through the vessel-wall, or through the small veins passing from the tympanic cavity to the bulb, cannot be determined.

That infection may occur directly through the wall will be understood after a study of the relations of the tympanum as shown in Figs. 1 and 2. In Fig. 1 the lines indicate openings in the wall of the tympanum near the floor. Fig. 2 shows the reverse side of the same bone with the openings leading directly into the jugular fossa. Here lies the jugular bulb and when, from any cause, there is a dehiscence in the bony wall the vessel is protected by thin mucous membrane only, and it can be readily understood how easily infection of the bulb may occur.

The two cases whose histories are given are clearly of the type indicated in the title of the paper.

Case I.—J. B., a male, aged 7 years. Family history good. Previous history negative. Immediately following an attack of measles he complained of severe pain in the left ear. When seen two days later the membrane was red and bulging, but the canal wall was apparently normal. Temperature 103° F. Patient very fretful. A very free paracentesis was followed by a considerable flow of blood and pus; the culture showed staphylococci. Polynuclear leukocytic percentage of eighty. Hot douching, hot compress, and calomel ordered. Marked improvement followed; temperature fell to 99.4° F. Patient was comfortable and quiet and remained so for 36 hours. Then, without the history of a chill, patient became restless and the temperature rose rapidly to 104° F. The probability of infection was considered and operation advised, but refused. The temperature fell gradually to 98.2° F., with marked improvement in the general condition apparently and this improvement continued for 76 hours, when again, without history of chill,

FIG. 1.—Showing perforations (*a*) in the bony tympanic wall through which infection may reach the jugular bulb directly. (From Randall's collection.)

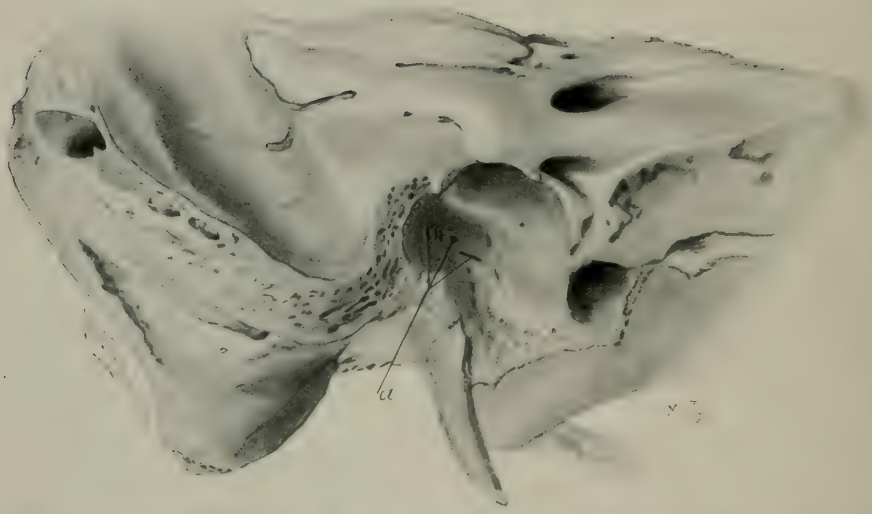

FIG. 2.—Showing perforations (*a*) into the jugular fossa in direct relation to the jugular bulb. (Same specimen as Fig. 1.)

the child became at first restless and irritable and later semi-comatose. The temperature rose to 106.2° F. The grave nature of the case was carefully explained to the parents and this time permission to operate was given.

Operation.—The mastoid was apparently perfectly normal, no pus or granulations being found until the antrum was entered. The sinus was then uncovered from the knee to its junction with the bulb. The upper part appeared normal, but the lower third was gray and partially collapsed. The internal jugular vein was now ligated below the entrance of the facial. Then the sigmoid sinus was opened and the infected clot removed from the bulb as thoroughly and carefully as possible.

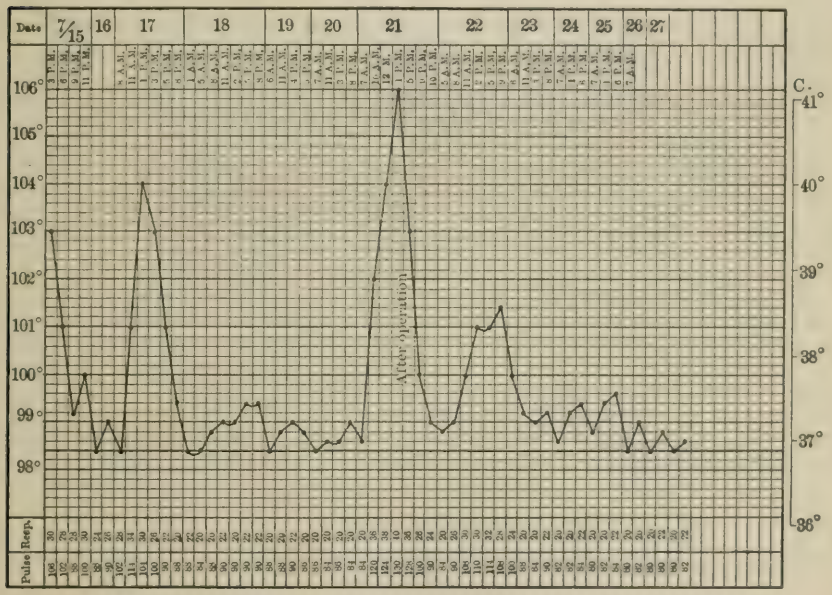

FIG. 3.—Temperature, pulse, and respiration in Case I.

There were two marked rises in temperature after the operation, but convalescence was practically uninterrupted and recovery complete with normal hearing. The clot gave a pure culture of staphylococcus. The chart (Fig. 3) shows the characteristic temperature.

CASE II.—S. W., a girl, aged 17 years. Family history good. Previous history negative. About three weeks before admission to the hospital she complained of pain in the right ear; this con-

tinued for a week without treatment, when there was a discharge from the ear, which persisted. One week before admission she complained of pain over the mastoid, with decrease in the amount of the discharge from the ear. On admission the patient was apparently suffering from some form of meningeal irritation; temperature 103° F., pulse 120. No history of chill. She was operated upon shortly after admission.

Operation.—The mastoid cells were found to be perfectly healthy, no pus or granulations being found until the antrum was

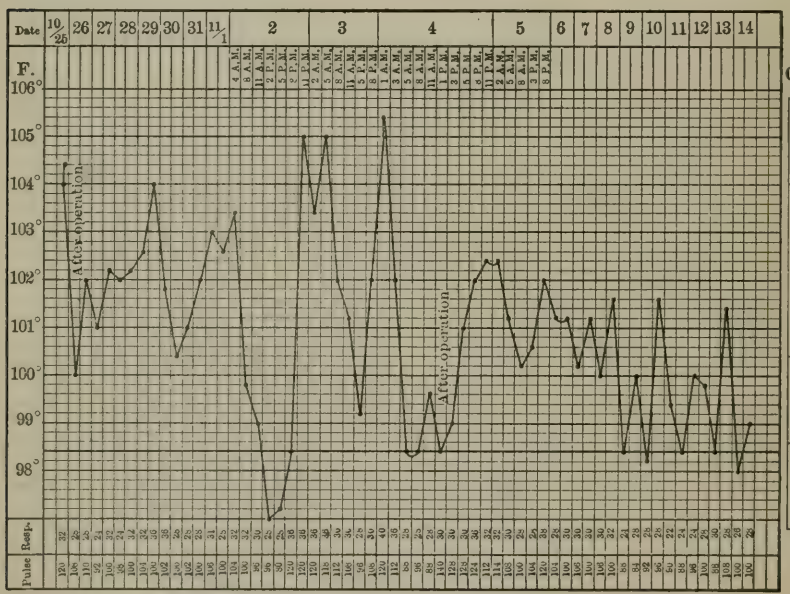

FIG. 4.—Temperature, pulse, and respiration in Case II.

entered, but here there was some pus apparently under considerable pressure. Culture showed staphylococci. The morning following the operation temperature had fallen to 100° F., and the patient was apparently improved. During the day she began to complain of intense pain in the forearms and legs. Dr. Harvey Shoemaker saw her in consultation and was of the opinion that she was suffering from an attack of acute rheumatism. As shown by the chart the temperature remained high and variable for about eight days following the operation; then without history of a chill there was a sudden fall of temperature to 97° F., and then a rise to 105° F.;

this occurred at 11 P.M. There was a polynuclear leukocytic percentage of 70 and leukocytosis of 15,000. The rapid and characteristic variations in temperature are shown by the chart (Fig. 4). It was decided to operate and examine the sinus.

Second Operation.—The granulations over the previously-operated surface were not healthy, but the underlying bone was. When the sinus was uncovered it was found to be gray and partially collapsed in its lower portion. The jugular vein was now ligated. Then the sinus was uncovered from above the knee to its junction with the bulb. The sigmoid portion, and also for some distance beyond the knee, was filled with cheesy pus. A flow of blood from the lateral sinus was easily established and this washed out the pus nicely. The external wall of the sigmoid sinus was excised and a well-formed clot found in the bulb.

Five days after the second operation pus was found in a large cavity over the sacrum. Two days later pus was found in both forearms in the cellular tissue extending from the wrist to the elbow; about 200 c.c. was evacuated from each arm and a large drainage tube passed from the elbow to the wrist. Two days later a collection of pus was found in each leg and about 300 c.c. was evacuated at the ankle and a drainage tube passed from the ankle to the knee. Subsequently pockets were found in the thighs, calves, below the scapula, and in various parts of the body, until 22 cavities in all were drained. The patient made a complete recovery.

The lesson that is so clearly taught by the above is that there is always grave danger of pyemic conditions arising in the very acute stages of middle-ear suppuration, and that we should be constantly on our guard and watchful for the signs of these conditions and not rest with a feeling of false security because the process is in an acute stage.

Pathology

LEUKEMIA AND SARCOMATOSIS

A LECTURE DELIVERED IN THE DEPARTMENT OF PATHOLOGY OF THE UNIVERSITY OF FLORENCE[1]

BY G. BANTI, M.D.

Professor of Pathologic Anatomy, and Instructor in Clinical Medicine and General Pathology, in the University of Florence, Chief Physician to the Hospital "Santa Maria Nuova," Florence, Italy

GENTLEMEN: The subject of the leukemias, in spite of the numerous works devoted to it, is still one of the most obscure in the domain of pathology. In this lecture, I purpose to lay before you the conclusions at which I have arrived after many years of study of the subject, conclusions which differ radically from the classical teachings.

The two theories most in vogue at the present time concerning the nature of the leukemias, are that of Ehrlich and his school, and that of Neumann, Pappenheim, Walz, Grawitz and others. Ehrlich, Pincus, Lazarus and others distinguish between two forms of leukemia, lymphatic and myeloid. In the first a hyperplasia of lymphatic tissue takes place in the lymph-glands and spleen, later also in the bonemarrow, with a secondary increase of the lymphocytes of the blood, producing a lymphocythemia. In the second variety the bonemarrow increases in amount, the spleen and lymph-glands also at times undergo myeloid transformation, both as to appearance and function. There is an increased formation of myelogenous, granular leukocytes, which reach the circulation in an immature state and are known as myelocytes; the cells are mononuclear, with eosinophilic, neutrophilic, and basophilic granules. They may produce a myelocythemia. Pincus in addition distin-

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guishes a lymphatic pseudoleukemia, which differs from ordinary leukemia in the small number of lymphocytes; these cases may change into leukemia at any time simply by an increase of lymphocytes in the blood-stream. Türk describes other cases under the name of "pseudoleukemia alymphemica," in which, in spite of a hyperplasia of the lymphatic tissues, there is no increase of lymphocytes in the blood.

Neumann, Walz, Pappenheim, Grawitz, and others maintain that all the leukemias are myelogenic in origin. They distinguish two kinds of tissue in bonemarrow, the lymphatic, morphologically and functionally similar to that of the lymphatic organs, and the medullary, the only really specific tissue of the bonemarrow. When the entire bonemarrow is affected, the myeloid form of leukemia results; when the lymphatic constituent alone is involved, the lymphatic type of leukemia develops. They admit that the hyperplastic stimulus may affect the spleen and lymph-glands first; the lymphocytes, being produced in excess, accumulate within the capsules of these organs, bringing about an increase in size and giving rise to lymphatic pseudoleukemia; as the lymphatic part of the bonemarrow becomes involved secondarily, the lymphocytes cannot accumulate on account of the unyielding nature of the bone, and are forced into the blood-stream, and leukemia results.

The following points are deducible from either theory, no matter which is correct: (1) The pathologic basis of the leukemias is a hyperplasia of the lymphopoietic and hematopoietic tissue; (2) the pathologic tissue is endowed with an increased formative power, the product being lymphocytes and granular leukocytes; and (3) the excessive number of white cells in the blood are either normal white corpuscles, or they differ from them by being in a state of immaturity (myelocytes).

I now desire to lay before you the results of my own investigations and shall begin with the discussion of lymphatic leukemia.

LYMPHATIC LEUKEMIA

The two forms of the disease, the acute and chronic, show certain slight morphologic differences, but none which cannot easily be explained by the difference in duration of the two forms.

Blood.—Two types are distinguished, according to the size of

the white cells: (1) With a predominance of white corpuscles having a diameter of from 6 to 7.5 μ; (2) with a predominance of corpuscles having a diameter of from 10 to 14 μ. The small cells have the morphologic and histochemical characters of normal lymphocytes; the larger ones are considered identical with the cells found in the germinal centers of normal lymphatic follicles. I cannot agree with this view, since the leukemic cells differ considerably, especially in regard to their nuclei from the germinal cells.

Associated with these cells, and sometimes even predominating over them, are found larger cells, 15 to 30 μ in size; they contain a single nucleus and possess a more or less basophilic, sometimes granular, protoplasm. They do not resemble ordinary lymphocytes very closely and cannot easily be confounded with them. Occasionally we may also find cells having the characteristics of endothelial cells; they may for a time even be numerous, but always disappear very quickly.

Nucleated red corpuscles are found constantly in acute leukemia, but they are very rare and may even be wanting in chronic cases.

Lymphatic Glands.—The enlarged lymphatic glands are not always movable and distinct from one another, but at times form one single mass, which may be adherent to neighboring organs; their capsules are more or less absorbed, or they may even infiltrate adjacent structures. Histologic examination shows the reticulum in some cases to be identical with that of normal glands, but frequently its filaments are much thicker and stronger. The character of the reticulum varies in the different cases, but in one and the same case is similar in all the affected organs. The greater number of the cells in the meshes are identical with those circulating in the blood; mixed with them are to be found either singly or in groups mononuclear cells, 15 to 30 μ in diameter; they are of an epithelioid character; also polynuclear cells, with a diameter of from 20 to 40 μ, having either separate or lobated nuclei. There are numerous intermediate types between the cells just mentioned. Many of the nuclei, especially those of the epithelioid cells, present characters of degeneration.

The lymphadenoid tissue found in the organs affected by the leukemic process is, in comparison with normal lymphadenoid tissue, atypical, especially as regards its cells, but often also as

regards its reticulum. This tissue develops first in the cords of the follicles, spreading from there to the follicles themselves, and finally invading the sinuses, gradually replacing the normal tissue. The follicles disappear passively and their germinal centers do not show any increase in productive activity; on the contrary, their cells present such phenomena of degeneration as karyolysis, karyorrhexis, etc. The connective-tissue trabeculæ, coming from the capsule, are then invaded by the lymphadenoid tissue, and finally the whole gland acquires a uniform type of structure without any distinguishable trace of follicles, sinuses, etc. Glands measuring from 2 to 3 mm. may be completely transformed. The leukemic lymphadenoid tissue may invade the capsules of the glands, and eventually extend to the surrounding tissues and organs.

The study of the blood-vessels coursing through this tissue is of great importance. Many of the blood-vessels, large in size, have walls formed of a single layer of endothelial cells. The walls of others are invaded by the lymphadenoid tissue, this penetrating as far as the endothelium, and occasionally projecting in the form of vegetations into the interior of the vessels. In some instances the walls are made up of lymphadenoid tissue, the endothelium having been completely destroyed. The cellular elements of this tissue are thus constantly entering the circulation. In spite of the lack of endothelium the blood does not coagulate in their interstices. The proof of the penetration into the circulation of the lymphadenoid cells is not hard to obtain: in the case of the bronchial glands cells containing coal particles are occasionally found with the other elements of the blood in the interstices of the blood-vessels.

Spleen.—The same changes described in connection with the lymph-glands are observed in the pulp, the follicles and the blood-vessels of the spleen, and even when the spleen is but slightly enlarged extensive alterations may be detected.

Bonemarrow.—This is of a reddish or reddish-gray color. This appearance may occasionally be met with throughout the entire skeleton; in other instances this type of marrow is scattered through the fatty marrow in the form of nodules. Some of these nodules may have a greenish color, as in chloroma. The bony tissue may be rarefied. Sometimes the bonemarrow appears as small reddish buttons beneath the periosteum, projecting through the dilated

Haversian canals; at other times the periosteum and its surrounding structures (connective tissue, muscles, etc.) are infiltrated with a grayish tissue similar to that of the lymphatic glands, and continuous with the bonemarrow.

In the transformation of the yellow into red bonemarrow the fat-cells become progressively smaller, and are separated by a homogeneous or slightly-striated substance, through which delicate capillaries with distinct endothelial walls course. The intercellular spaces are then invaded by the elements of the blood, thus forming large intercommunicating lakes of blood, which are devoid of a proper wall and are bordered by the membrane of fat-cells. Later, within these lakes of blood and around the blood-vessels, appear islets made up of functionating bonemarrow (containing myelocytes, megalocytes, nucleated red corpuscles, etc.) Later still nodules of lymphadenoid tissue similar to that of the glands and spleen develop around the small arterioles, and increase in number until finally the entire bonemarrow is transformed completely. The same alterations in blood-vessels as described with reference to lymphatic glands and spleen occur in the bonemarrow.

These changes may be divided into four stages:

(1) The hyperemic or telangiectatic stage;
(2) The hematopoietic stage;
(3) The nodular lymphadenoid stage; and
(4) The diffuse lymphadenoid stage.

In acute leukemia death may supervene in the first or second stage; more commonly it occurs in the third, and rarely in the fourth stage; the latter is more characteristic of chronic leukemia.

These alterations explain very well why in acute leukemia nucleated red cells in the blood are the rule, whereas in chronic leukemia they are rare or wanting; they also explain the occasional presence in the blood of myelocytes in lymphatic leukemia. These investigations also seem to establish beyond a doubt, and contrary to the opinions of Neumann, Pappenheim, and others, that lymphatic leukemia may occur without lymphadenoid transformation of the bonemarrow.

In addition to the alterations already described, there are others more inconstant; they consist of lymphadenoid nodules or infiltra-

tions in different organs. The more important ones are in the following tissues:

Serous Membrane.—The pleura and the pericardium are the seat of more or less numerous lymphadenoid nodules, similar to those found in the lymphatic glands and the spleen. In some of the cases the nodules are few and disseminated, in others so closely grouped as to suggest confluent tuberculosis. The nodules are uniform in size and in general larger than tubercles. In other cases these membranes are the seat of a uniform infiltration, particularly when a large lymphadenoid tumor in their neighborhood has spread to them by contiguity. (From the thymus, for instance.)

Liver.—The lymphadenoid tissue always develops in the portal spaces. It may occur in the form of small isolated nodules in the neighborhood of the interlobular branches of the hepatic artery, but more frequently, and particularly in acute leukemia, it develops around the interlobular branches of the portal vein, and is uniformly diffused through the interacinous spaces. This diffuse infiltration is found only in cases in which the spleen or intestines share in the morbid process.

Kidneys.—The same alterations may be present in the kidneys. In the diffuse form both kidneys may be equally involved, the cortex being especially affected. The renal tubules are surrounded by a uniformly-sized cylinder of lymphadenoid tissue.

Intestines.—In addition to the well-known tumefaction of the normal lymphatic nodules of the intestinal mucosa, lymphadenoid nodules are met with around the arterioles between the two layers of the muscular coat.

Lungs.—Here I have invariably met with the lymphadenoid nodules; they develop in the small lymphatic follicles described by Arnold.

Tonsils.—These organs, if affected, may present a structure similar to that of lymphadenoid tissue elsewhere. More frequently however the intercellular connective tissue is made up of scanty, irregularly-arranged fibers, as in the ordinary round-cell sarcomas, and does not resemble cytogenous tissue.

The lymphadenoid alterations of the serous membranes, liver, kidney, intestines, and lungs, are to be regarded undoubtedly as secondary. The generally-accepted opinion as to the mechanism of

their production is the theory that we deal with a hyperplasia of pre-existing lymphatic tissue (Ribbert, Waltz, Grawitz, etc.). While this may be true for some organs, such as the nodules of the lungs, it cannot be true for all cases. The innumerable nodules occasionally seen on the serous membranes cannot be attributed to such a hyperplasia, as no observer has ever been able to discover lymphatic tissue there under normal conditions. Neither is it possible to attribute to hyperplasia the abundant lymphadenoid tissue so uniformly found in the liver around all the branches of the portal vein. The preëxistence of normal lymphatic tissue all over the liver is made the more improbable, as ordinarily it disposes itself about arteries and here it surrounds veins. The same is true of the lymphadenoid tissue which surrounds the tubules of the kidneys, that which is found in the muscular layer of the intestines, or in the myocardium, and other muscles. It must also be recalled, that the periportal new formation is found only when the spleen or intestines are involved; when these organs are not affected, the alterations assume the aspect of nodules around the branches of the hepatic artery.

These facts, and others omitted for the sake of brevity, prove irrefutably that many of the secondary lymphadenoid new formations are true hematogenous metastases. Lymphadenoid elements, which have become detached from the organs affected by the leukemic process and conveyed to distant parts, are able to develop at a later stage into secondary colonies of a tissue similar to the primary ones, even in parts in which normally no lymphatic tissue existed. The property of producing metastases indubitably brings the leukemias closer to the true neoplasms. Another feature in which leukemia resembles neoplasms is that leukemic lymphadenoid tissue may break through lymphatic glands or bone and invade and extensively infiltrate neighboring tissues.

According to the classical theory the leukemic products in the lymphatic glands, spleen, etc., are also due to hyperplasia of normal lymphatic tissue. A number of reasons, the most important of which I wish to mention, make this view untenable:

(1) The structure of the leukemic lymphadenoid tissue is often atypical, owing to the character of both reticulum and cells.

(2) The leukemic lymphadenoid tissue takes the place of the

normal reticulum in spleen and lymph-glands; the follicles atrophy and disappear without presenting signs of increased formative activity.

(3) The leukemic lymphadenoid tissue regularly invades the trabeculæ, and frequently also the capsules of the lymphatic glands.

(4) This tissue may also break through the borders of the lymph-glands to invade adjacent tissues.

(5) The walls of the blood-vessels, particularly of the veins, are constantly invaded.

(6) The leukemic deposits frequently break into the interior of the blood-vessels.

(7) Hematogenous metastases are constantly found.

(8) In some organs invaded by the leukemic process the new intercellular tissue may differ from the normal reticulum.

(9) In the bones there may be rarefaction, owing to the erosion produced by the new-formed lymphadenoid tissue.

(10) After rarefying the bone, the lymphadenoid tissue may attack the periosteum and the paraosteal tissues.

The name "hyperplastic" should not be conferred on a tissue with atypical structure, which shows a tendency to invade neighboring tissues and gives rise to metastases. Such a tissue should be spoken of as neoplastic rather than hyperplastic. Hence lymphatic leukemia must in my opinion be classed with the neoplasms, or to be more explicit, must be considered to be *a systematic lymphadenoid sarcomatosis of the lymphopoietic and hematopoietic organs.* To support this opinion I am able to adduce still other arguments:

(1) In certain sarcomas we frequently meet with the blood-vessel alterations described above, that is, the blood circulates in channels deprived of their endothelium and bound by sarcomatous tissue.

(2) In sarcomas there is frequently an increase of mononuclear cells in the blood resembling lymphocytes. Many authors speak of lymphocytosis in these cases, but in reality we are dealing with neoplastic cells from the tumor.

(3) Occasionally true lymphatic leukemia is found associated with sarcomas which have remained localised. Post-mortem examination however reveals an invasion by the sarcoma of the interior of a larger vein. The case reported by Luecke is very convincing in this respect.

(4) Between the cases of typical lymphatic leukemia, in which the lymphatic glands remain quite distinct and the leukemic tissue does not break through the capsule, and the cases in which groups of lymphatic glands fuse and neighboring organs are extensively infiltrated with the newly-formed tissue, there exist innumerable intermediate cases, which differ so little from the preceding and the succeeding type as to make it quite clear that all belong to one and the same morbid class.

(5) Although chloroma has occasionally instead of a lymphadenoid texture that of a round-cell sarcoma, it is frequently associated with a leukemic increase of mononuclear cells (the lymphatic leukemia of some writers).

(6) The same is true of another form of systemic sarcomatosis, *multiple myeloma* or *Kahler's disease.*

Lymphatic leukemia, whether acute or chronic, is therefore according to my opinion a lymphadenoid sarcomatosis. The sarcomatous tissue invades the blood-vessel walls, destroys the endothelium, and finally penetrates the blood in variable quantities. As long as the endothelium is intact, we have to do with *pseudoleukemia alymphemica* (*Türk*). When the endothelium begins to be destroyed, and small numbers of sarcoma cells enter the circulation, we are dealing with *lymphomatosis sublymphemica* (*Türk*), or *lymphatic pseudoleukemia* (Pincus). When large numbers of cells enter the circulation, we are dealing with *lymphatic leukemia.*

As proof of the fact that sarcoma cells enter the circulation after destroying the endothelial lining, I had a case in which the leukocytes suddenly increased from 23,000 to 280,000 per c.mm.; this increase coincided with the appearance in the blood of a considerable number of endothelial cells, which the next day had almost completely disappeared. A leukemic focus had evidently ruptured the endothelium and entered the circulatory stream.

By the sarcomatous hypothesis all the pathologic and clinical facts heretofore unexplainable can be readily accounted for, viz:

(1) The morphologic differences between leukemia and pseudoleukemia;

(2) The fact that pseudoleukemia may unexpectedly be transformed into leukemia.

(3) The daily oscillations, sometimes very considerable, in the number of circulating white cells.

(4) The fact that leukemia may change into pseudoleukemia; (explained by thrombosis of the affected vessel).

(5) The fact that with an unexpected increase in leukocytes, a diminution in the size of the lymphatic glands is occasionally observed; this is due to their contents emptying into the blood-vessel.

(6) The fact that leukocytes in the blood, erroneously considered lymphocytes, are at times so atypical, as to make it uncertain, whether they are really lymphocytes, or probably of different origin —myelocytes.

The resemblance between lymphatic leukemia and sarcoma has been pointed out for years by such pathologists as Bignami, Strauss, Drozda, Türk, and others; Drozda went the farthest in this direction, when he published under the name of "Sarcomatosis Leukemica" a case which he represents as a transitional link between leukemia and sarcoma. According to my view, these authors grasp but a part of the truth: *Lymphatic leukemia is a unique disease; it is always alike in its nature, and is a special form of sarcomatosis.*

MYELOGENOUS LEUKEMIA

This form is almost always chronic, though acute cases undoubtedly occur.

Blood.—The prevailing white cells in the circulation are mononuclear with or without granulations (neutrophilic, eosinophilic, or basophilic) in their protoplasm; they are the myelocytes of Ehrlich.

I distinguish two types of blood according to the size of the white cells: (1) With cells having an average diameter of from 12 to 15 μ; and (2) with cells having a diameter of from 18 to 25 μ.

Cells of a diameter of from 30 to 40 μ and even more may be found in both types in variable and sometimes even excessive number; sometimes cells without granulations predominate; in other cases there are also multinuclear giant-cells or their nuclei. The occurrence of nucleated red corpuscles in the circulation is well recognized.

Bonemarrow.—Generally it has about the same structure as the

normal marrow, but upon closer study it shows a number of variations. The cells are generally larger, and their granulations more variable in number and size. The giant-cells vary much in number and dimensions; in general they are smaller, though at times larger than normal; their nuclei are multiple rather than lobulated. In some cases the reticulum, instead of having the usual delicate appearance, is made up of thick ribbon-like fibers. The cells are few in number and distributed through the meshes of this coarse reticulum.

Changes in the blood-vessels similar to those described for lymphatic leukemia constantly occur. The myeloid tissue shows a tendency to invade the blood-vessel walls, occasionally forming small tumors, which lift up the endothelium and project into the interior of the vessels; the endothelium may be broken through, the vegetations coming in direct contact with the blood. The large blood-spaces bordered by the cells of the myeloid tissue are also met with, their cells mixing with the blood; thromboses in these vessels have never been noted.

New formation of osseous tissue may occur in the medullary canal, at times almost obliterating it. In other cases the myeloid overgrowth may produce rarefaction of the bone. Rarely the periosteum and the paraosteal tissue may be infiltrated by the myeloid tissue.

Lymphatic Glands and Spleen.—When these organs are involved by the disease, the lymphatic tissue is replaced by myeloid tissue which reproduces the structure of the bonemarrow. This substitution begins at a number of points and gradually spreads over the organs; their elements atrophy and degenerate without taking an active part in the new formation; the organs may be completely transformed into myeloid tissue. Each irradiation center of myeloid tissue is invariably formed by a blood-vessel. Exceptions to this rule may occasionally be noted in the lymphatic glands. When individual glands in the same group become successively affected in the direction of the lymph-current, the myeloid transformation begins from the peripheral lymphatic sinuses and is not connected with the blood-vessels. Their blood-vessels are subject to the same alterations as those of the bonemarrow. Small glands not over 2 mm. in diameter may be entirely transformed into myeloid tissue.

There is no doubt that the bonemarrow must be looked upon as the primary seat of the disease. In order to explain the genesis of the secondary foci, they must be studied from their inception and followed throughout their evolution. Thus it is easily shown that the organs secondarily invaded remain completely passive, that their cells degenerate and disappear, and that the new myeloid tissue is derived entirely from myeloid elements brought by the blood. The elements proliferate, producing at first nodules and afterward infiltrations. The polynuclear giant-cells in the secondary foci are not hematogenous in origin, but are derived by progressive evolution from the mononuclear cells which have migrated from the blood-vessels. The secondary foci are therefore metastatic, and the myeloid cells must be considered similar to tumor cells capable of inducing metastasis. In the lymphatic glands, as before noted, metastases may occur by the lymphatic route; the myeloid cells are arrested in the sinuses.

Of the many arguments militating against the hyperplasia theory of the production of the myeloid tissue in myelogenous leukemia, the following are the most important:

(1) The myeloid tissue, both primary and secondary foci, has always a more or less atypical structure as compared with normal bonemarrow.

(2) The myeloid tissue invades the blood-vessel walls, particularly of the veins, and comes in direct contact with the blood by destroying the endothelium. The presence of blood-channels without walls is characteristic of sarcomas.

(3) The myeloid cells produce hemotogenous and lymphogenous metastases, just as do the cells of malignant neoplasms.

(4) The metastatic nodules, as in cancer, are produced by proliferation of important specific cells; the elements of the organs involved take no part in their formation.

(5) The osseous tissue may undergo eburnation or rarefaction as in osteosarcoma.

(6) The periosteum and surrounding tissues may be involved by the extension of myeloid tissue from the bones.

(7) The mononuclear leukocytes (myelocytes of Ehrlich) found in the blood are often atypical; they may be very large, in comparison with myelocytes of normal bonemarrow.

These facts manifestly favor the neoplastic theory of myelogenous leukemia; it should be included among the sarcomas, and more particularly classed with the group which arises from bone-marrow; it reproduces its structure even to the nucleated red corpuscles.

According to this view myelogenous leukemia is a *systematic myeloid sarcomatosis of the hematopoietic and lymphopoietic organs.* The so-called myelocytes are neoplastic cells which have entered the circulation owing to the changes in the blood-vessels described above.

According to my experience there exists, corresponding to the lymphatic pseudoleukemia, a *myeloid pseudoleukemia.* It is characterized by more or less intense anemia, and the presence in the blood of a small number of Ehrlich's myelocytes. The number of leukocytes in the blood is approximately normal, but later the blood acquires the typical character of myelogenous leukemia—sometimes gradually, sometimes suddenly. In the preleukemic period splenomegaly is sometimes wanting. There is a myeloid transformation of the bonemarrow and sometimes of the spleen, but the communication between the new-formed tissue and the interior of the vessels is wanting. When this change occurs the disease changes to leukemia.

From these facts I hope that you are convinced that the leukemias are not hyperplasias of normal tissues, but diseases of a neoplastic nature, belonging to the vast and proteiform class of sarcomas. I am not able to explain their etiology, though I am convinced that they are infectious sarcomatoses, but heretofore all my researches to discover the specific agent have had a negative result.

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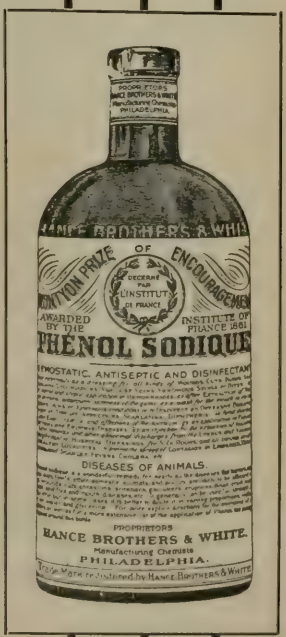

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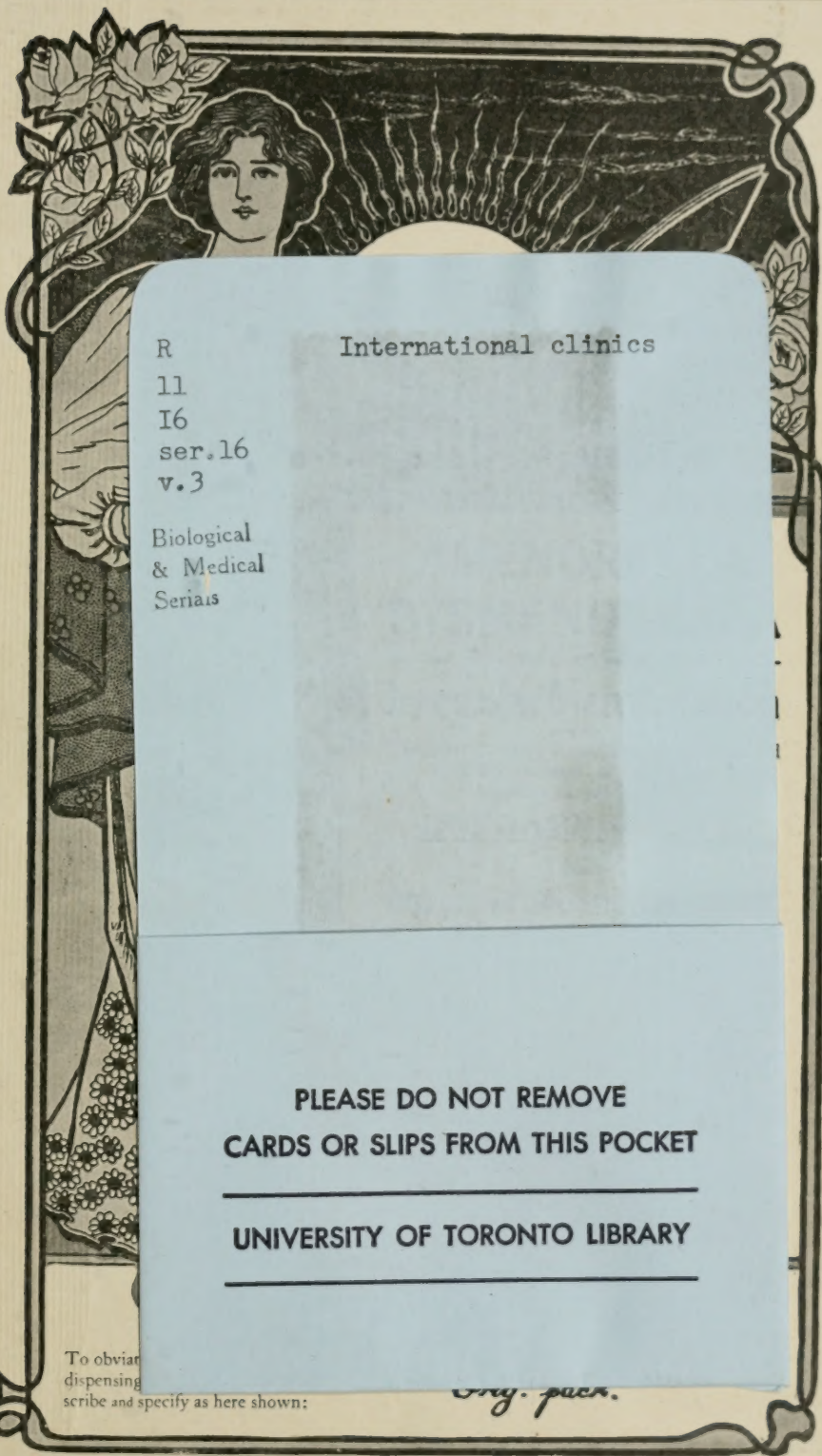

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